DISSERTATION

on

CORRELATION BETWEEN SARCOPENIA AND VITAMIN D LEVEL IN ELDERLY POPULATION

submitted in partial fulfillment of

requirements for

MD DEGREE EXAMINATION

BRANCH - XVI GERIATRIC MEDICINE

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI

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CHENNAI – 600003

APRIL 2015

CERTIFICATE

This is to certify that the dissertation titled **"CORRELATION BETWEEN SARCOPENIA AND VITAMIN D LEVEL IN ELDERLY POPULATION"** is a bonafide work done by **Dr.S.PRABHAGARAN**, Post Graduate Student, Department of Geriatric Medicine, Madras Medical College, Chennai – 600003, in partial fulfillment of the university rules and regulations for the award of MD DEGREE in GERIATRIC MEDICINE BRANCH - XVI, under our guidance and supervision, during the academic period from April 2012 to April 2015.

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DECLARATION

I solemnly declare that the dissertation titled **"CORRELATION BETWEEN SARCOPENIA AND VITAMIN D LEVEL IN ELDERLY POPULATION"** was done by me at Madras Medical College, Chennai – 600003, during the period June 2014 to August 2014 under the guidance and supervision of **Prof. S.SIVAKUMAR, MD, DTRD,** to be submitted to The Tamil Nadu Dr. M.G.R. Medical University towards the partial fulfillment of requirements for the award of MD DEGREE in GERIATRIC MEDICINE BRANCH-XVI.

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CONTENTS

ABSTRACT AND KEYWORDS

TITLE: CORRELATION BETWEEN SARCOPENIA AND VITAMIN D LEVEL IN ELDERLY POPULATION

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BACKGROUND:

Sarcopenia is defined as progressive reduction in musclemass, muscle strength and function that affects older people.Muscle mass decrease is probably the single most frequent cause of late-lifedisability among older people. It is directly responsible for functional impairment with lossof strength, and increased likelihood of falls and fractures.Many studies have shown that low serum 25(OH)D levels are related to accelerated losses in muscle mass and reduced muscular strength, reduced muscle power and gait speed. In this study relation between vitamin D level and sarcopenia was analyzed.

METHODS:

50 elderly patient of age more than 65years without sarcopenia and 50 elderly with sarcopenia in Geriatric department were selected. Elderly with sarcopeniawere selected based on EWGSOP definition using muscle mass, strength and gait speed.The vitamin D level of both the group were compared.

RESULTS:

Prevalence of vitamin Ddeficiency(includes both deficiency and insufficiency) in both group were 78% and 86% respectively. Mean Vitamin D levelin both groups were 41.03nmol and 58.69nmol respectively. Severe deficiency with vitamin D less than 25nmol was seen in 12% and 6% in sarcopenic and non sarcopenic elders respectively.

CONCLUSION

Vitamin D deficiency is one of the contributing factors for sarcopenia and the prevalence rates for severe vitamin D deficiency is higher in sarcopenic elderly

KEYWORDS

Sarcopenia Vitamin D, Deficiency

INTRODUCTION

Musculoskeletal system is one of the important organ system in the human body. The disease and disorder ofmusculo skeleton are common in late life and have a major influence on function and quality of life. There are many disease that affect the musculo skeleton and are forms of myopathy.

The disease or condition that cause loss of skeletal muscle mass is called sarcopenia. The wordSarcopenia derives from the Greek and means “poverty of flesh.” In medical terms, sarcopenia is defined as a nonspecific and can be caused by aging, wasting, disuse, illness, or starvation or can also a secondary consequence of neuropathy or ischemia.Sarcopenia is more common in older populations. There are multiple contributing factor for sarcopenia that includes, early life developmental influences, the ageing process over the life course, less than optimal diet, sedentary lifestyle style or bed rest, chronic illness, certain drug treatments and hormonal influence. Sarcopenia represents an impaired state of health with many adverse outcomes that includes

- mobility disorders
- disability
- impaired ability to perform ADL

- increased risk of falls and fracture
- loss of independence
- reduced quality of life and
- increased mortality.

Vitamin D, which is a fat soluble vitamin and a major steroid hormone has a major role in bone and calcium metabolism. Apart from this function, it also plays a major role in cell growth regulation, bone formation, strengthening of skeletal muscle, regulation immune system, muscle strength, hair growth, immunity against infection, reducing the risk of autoimmune diseases, role in systemic hypertension and diabetes mellitus.

Some of the morphological changes in musculo skeletal system was associated with vitamin D deficiency. For example, patients with osteomalacia with muscle dysfunction associated with vitamin D deficiency show degenerative changes such as fat infiltration in muscle fibres, opaque fibers, ghostlike necrotic muscle fibers, regenerating andfibrosed muscle, enlarged interfibrillar spaces, fibrosis, glycogen granules, andatropy of the type II muscle fiber**.** As is the case with vitamin D-deficient patients, it is well known that elderly people show aberrant muscle morphology. Many recent studies shows that vitamin D deficiency as one of the contributing factor for progression of sarcopenia. Also many interventional study showed

a significant improvement in muscle strength by vitamin D supplementation in elderly population. Vitamin D deficiency is more prevalent in elderly. Older individual with poor dietary intake, chronic bedridden, stays indoor will have subclinical vitamin D deficiency. Vitamin D deficiency in older people is thought to occur mainly due to decreased exposure to sunlight, poor dietary intake of vitamin D, and reduced skin capacity of the skin to synthesis vitamin D.

However there are no Indian studies on correlation between sarcopenia and Vitamin D level. Hence an attempt to assess the correlation between vitamin D level and sarcopeniawas done in this study.

AIM OF THE STUDY

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1. To assess the correlation between sarcopenia and Vitamin D level in the elderly population.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Practice of Geriatric medicine mainly focus on older population who are frail, as they are thought to be the population at highest risk for various adverse outcomes [1] Although in elderly the physiological ageing changes may be the foundation of frailt , the phenotype of frailty initially present as loss of weight or loss of muscle mass (sarcopenia) even before the establishment of adverse outcomes. This results in weakness of muscles, decreased exercise tolerance or endurance, low physical performance and slowed task performance (such as gait speed)[2–4]

PHYSIOLOGY OF SKELETAL MUSCLE:

All skeletal muscle are enclosed by a sheath of connective tissue called epimysium. Bundles of muscle fibers and individual contracting units of muscle fibers are surrounded by partitions of the connective tissue constituting the perimysium and the endomysium, respectively. Blood vessels, lymphatics, and nerves reach all the muscle compartments by way of these fibrous partitions. Matured single muscle fibers are long multinucleated and cylindric units separated from other cells and a membrane surrounding this unit, the sarcolemma. Skeletal muscle fibres contain myofibrils which is made up of the contractile proteins myosin, actin, tropomyosin, and troponin and the structural supportive proteins actinin and titin. The particular spatial

arrangement of the thick and thin contractile proteins is responsible for the characteristic cross-striations in skeletal muscle.[5]

MOTOR UNIT:

Lower spinal cord motor neurons provide a common pathway for transmitting neural impulses from upper motor levels of the central nervous system to the skeletal muscle. This information is directed to skeletal muscles via the ventral roots, peripheral nerves, and cranial nerves. Within the single muscle the muscle fibers are innervated by motor neurons and these motor neurons are grouped in the spinal cord as it innervates the single muscle. This forming the motor neuron pool for that muscle. The activation of a motor neuron brings all the muscle fibers to the mechanical threshold, therefore, a single motor neuron and its associated muscle fibers constitute the motor unit that is the smallest that can be activated to induce movement. On the basis of the speed of contraction, three types of motor units can be distinguished:

- fast-fatigable motor units,
- fatigue-resistant motor units
- slow-motor units (which is in between the two subtypes in terms of fatigue initiation)

These differences in motor unit performance are based on physiologic and biochemical characteristics of their constituent muscle fibers.[6]

CONTRACTION:

The raised intracellular calcium concentration is the vital event in muscle contraction which occurs due to the transduction changes in sarcolemma. Muscle relaxes as a result of calcium pumping back to the SR, release of calcium from troponin, and cessation of interaction between actin and myosin filaments.

NEURAL:

Several factors regulate muscle contractions, including innervations of muscle fibers. Muscle fibres innervation state alteration leads to change in the expression of gene which transcript protein involved in the excitation – contraction coupling. This variation in expression leads to excitation – contraction coupling difference. During development, the spinal motor neuron innervates the muscle fibers. This various subpopulation of motor neuron determines the fiber type by activating the contraction at various rates Fiber type is determined by interaction with different subpopulations of these motor neurons that activate contraction at different rates, ranging from 10 Hz (slow twitch) to 100 Hz (fast twitch fatigue resistant) or 150 Hz (fast twitch fatigue sensitive). Depolarization of myotubes in culture stimulates the

appearance of DHPR (dihydropyridine) binding sites, suggesting that DHPR expression was induced by the induction of muscle activity during innervation[7,8]

AGEING AND MUSCLE:

40–50% of the human body was mainly comprised of skeletal muscle and the skeletal muscle is composed of muscle tissue, connective tissue, blood vessels and nerves. Bundle of muscle fibers formed from the fusion of precursor cells called Myoblasts. The muscle fibers is of two types namely

1) Type I or slow twitch

2) Type II or fast twitch.

Type II muscle fiber consists of two components:

1) type A are called fast-oxidative fibres.
2) type B are known as fast-glycolytic fibres.

The normal ageing process shifts the type of fibre towards TypeI muscle fibre.[11]. Muscle contraction is of various types

- Isometric contraction
- Shortening contractions,
- Lengthening contractions.

The adaptive response of the skeletal muscles is to protect themselves from the damage caused by ROS – reactive oxygen species. Sarcopenia – the reduction in muscle mass with ageing that lead to decline in muscle strength and muscle power. After 50 years of age there will be decline in basal metabolic rate (BMR) by 4% per year. The myosin heavy chain production also reduces with age but the sarcoplasmic protein pool remains unchanged [12, 13]. The ability of the skeletal muscle to regenerate and hence the overall muscle mass decrease with age. The infiltration of fat into the muscle also increases with ageing. Many ageing changes occurs due to decline in physical activity. During ageing there will be loss of muscle fibres and motor neuron. The loss of muscle fibres is mainly due to the loss of motor neurons. The ageing muscle shows a decline in motor unit demonstrated by many electrophysiological studies[14]. With ageing, the size of the average motor unit increases with age. In some muscles, the limitation in the maximum force generated in older individual is not due to the failure of the central nervous system but it is mainly due to the decline in firing of the individual motor nerves at the time of maximal voluntary contraction [15]. The

fatigability of the musclederiug a sustained contraction is common in elderly compared to younger persons. Atropy of the ageing muscle fibre is more restricted towards type II muscle fibre this selectivity is more important for the muscle performance because the type II muscle fibre generate more power compared to type I especially in leg muscles. The relation between the rate of muscle contraction and muscle relaxation to the dysfunction in the calcium release is still not fully understood. Welle*et al*. study illustrate that aged muscle decreased expression of mRNA that encodes protein which plays role in ATP synthesis and miticondrial electron transport chain [16]. With the ageing process there will be more accumulation of DNA deletion in the miticondria of the skeletal muscle [17]. Thus during senescence the changes in the skeletal muscle affects muscle bulk, muscle performance and strength, endurance and function.

In the older individual, the loss of functional independence and the disability is contributed by the ageing changes of the skeletal muscle. Age-related weakness may be associated with fatigue, weakness discerned as an absolute decrease in muscle force, and fatigue as a progressive decline in force with prolonged physical activity. Decreased in muscle strength is associated with decreased in ADL like climbing stairs, getting up from the chair that leds to loss of functional independence.

AGE RELATED CHANGE IN MUSCLE A SUMMARY:

1. Reduction in muscle mass (30–40%)
2. Decreased myosin heavy chain synthesis
3. Decrease in force
4. Infiltration of fat into muscle tissue
5. Increased fatigability
6. Decrease in basal metabolic rate
7. Decreased innervations
8. Increased number of myofibril per motor unit
9. Loss or reduced proliferation of satellite cells
10. Shift towards type I fibers

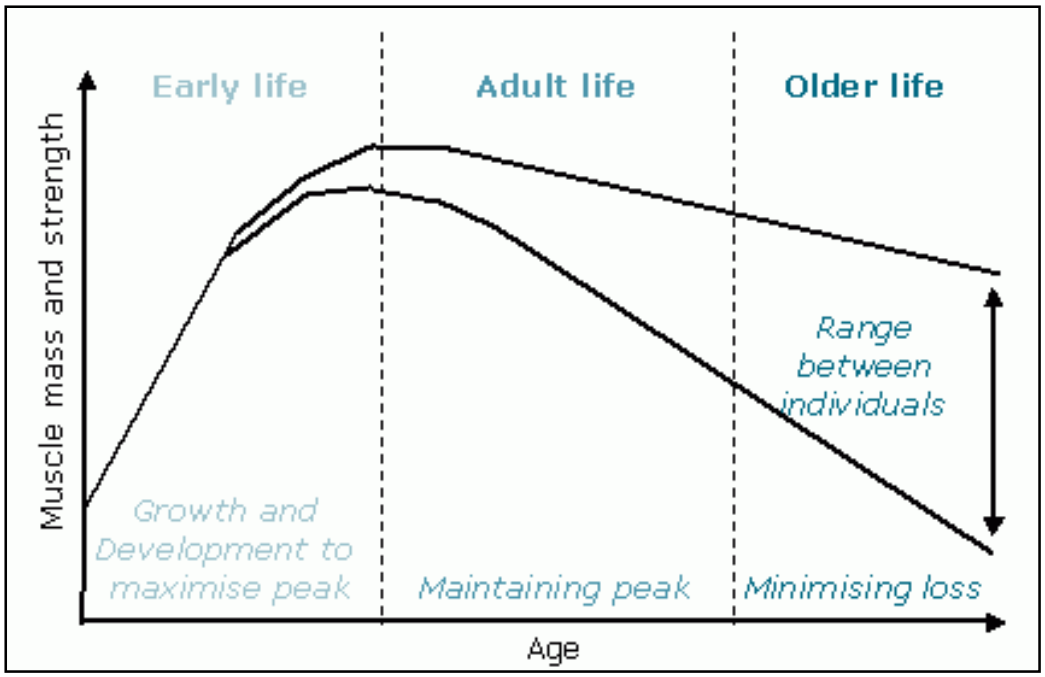


FIG.1 AGEING DECLINE OF MUSCLE MASS AND STRENGTH

CONTRIBUTION FOR WEAKNESS IN AGEING:

NEUROGENIC FACTOR:

Neurogenic mechanisms of decline in muscle function with age include reduction in the number and/or size of spinal cord motor neurons, alterations in axonal flow, and the neuromuscular junction. Each of these factors, individually or in combination, leads to chronic muscle denervation and motor unit remodeling and hence contribute to the weakness in ageing.

MYOGENIC FACTOR:

Primary muscular or myogenic factors refer to a group of alterations including contraction-induced injury and alterations in muscle signal transduction (trophic factor/ hormone resistance). The phenomenon of contraction induced injury is related to increased mechanical frailty and decline in muscle restorative capacity with age. Also, older muscles recover more slowly and do not exhibit complete recovery when compared with those in younger population. Eccentric contraction injury has also been demonstrated in older individuals. Insulin and IGF-1 resistance in aging skeletal muscle have been reported that further contributes to the progression of sarcopenia.

COMBINED FACTORS:

Combined mechanisms include muscle unloading and EC uncoupling. Muscle unloading associated with sedentary lifestyle is a major determinant of muscle atrophy in the elderly population. The decrease in physical activity is a combined process in which the lower the nerve activation, the lower the muscle contraction. This process leads subsequently to muscle atrophy.

Apart from nutritional, immobility, sedentary lifestyle, malnutrition and other contributing factor these neurogenic and myogenic factor of ageing

process further add up to the pathophysiological of sarcopenia. These factor increase the progression and worsening of sarcopenia.

Even though the ageing muscle contributes to the progression of decline in muscle strength there are many other contributing factor which further progress the sarcopenia.

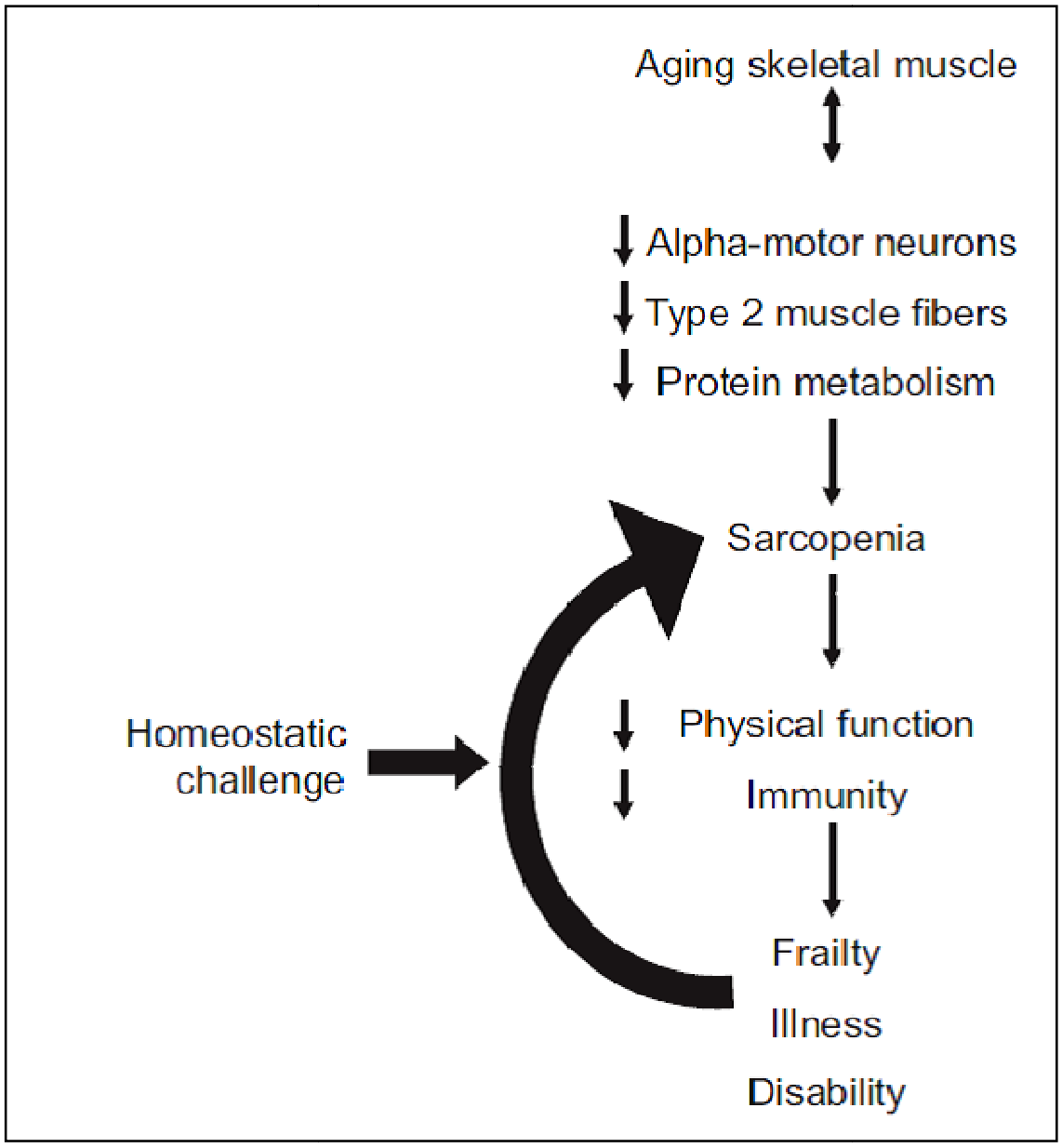


FIG.2 VICIOUS CYCLE OF SARCOPENIA AND DISABILITY

SARCOPENIA:

The capacity to live independently is essential for quality of life at all ages. With ageing this capacity is endangered by the degeneration seen in elderly overlying by co morbid illness. Decline in skeletal muscle strength and performance is one of the major determinant for functional independent apart from cognitive decline in elderly. In the past decade the annihilating effect of osteoporosis and dementia have been concentrated, but the sarcopenia that include decline in muscle strength and performance which is the cornerstone for frailty have not been focused. In 1989, Rosenberg [18]defined the term sarcopenia that refer to the age related loss of skeletal muscle mass and size. The word sarcopenia is formed from the greek word ‘sarx’which means fleshand the word‘penia’ which means loss. Rosenberg’s principle for defining the term sarcopenia that encapsulated ageing decline in muscle mass as much importance has not been given that time. The phenomenon of loss of muscle strength and even muscle mass with ageing was observed years earlier in 1931 when Critchley [19] noted that muscle mass loss occurs with ageing and the process of loss of certain fibre type in human skeletal muscle over time was observed in studies of muscle biopsies even after the first decade of life [20]. The decline in skeletal muscle with ageing was around 6% every decade which is showed by many longitudinal studies. The decline in muscle mass starts around 45 years of age. Therefore a

typical 85 year old person will have a muscle mass and size approximately three quarters of that when he or she was at 45 years of age. Even elderly population who are functionally independent, active and healthy are not immune to the sarcopenic process and it seems as though everyone loses muscle mass as they grow older [21]. Thus, as per Rosenberg's definition the occurrence of sarcopenia in elderly population is 100%. With that being said there are appreciable difference from person to person in peak skeletal muscle mass. Because of these tremendous difference there is variation in the muscle mass value of various elderly. Some of the older adults have a muscle mass comparable to healthy young adult, whereas other have a muscle mass so low that their functional ability and capacity severely compromised. Though the concept of saropenia is barely more than 20 years old it has gained popularity in academic circles and clinical setting in the west.

Geriatric syndromes are due to the unknown interaction between disease and ageing changes that results in producing a constellation of signs and symptoms. Geriatric syndrome leads to involvement of multiple systems. Instability, falls and incontinence are some of the geriatric syndromes [22].We suggest Sarcopenia to be considered as a Geriatric syndrome because this will promote the treatment and management of sarcopenia[23 – 24]**.**

DEFINITION:

Sarcopenia is a syndrome characterised by progressive and generalised loss of skeletal muscle mass and strength with a risk of adverse outcomes such as physical disability, poor quality of life, increased morbidity, low functional independence and death [25, 26].

The European Working Group on Sarcopenia in Older People (EWGSOP) defined a clinical definition and diagnostic criterion for sarcopenia.

The EWGSOP included representatives from four participant organisations

1. European Union Geriatric Medicine Society (EUGMS),
2. European Society for Clinical Nutrition and Metabolism (ESPEN),
3. International Assoc. of Gerontology and Geriatrics –European region
4. International Association of Nutrition and Aging.

The EWGSOP recommends the use of both skeletal muscle mass and muscle function (strength or performance) for the diagnosis of sarcopenia. The diagnosis of sarcopenia needs the presence of criteria 1 and the presence of either criteria 2 or 3. Diagnosis of severe sarcopenia is based on criterion 1 plus criterion 2 plus criterion 3 [27].

CRITERION FOR DEFINING SARCOPENIA:

1. Low muscle mass

2. Low muscle strength

3. Low physical performance

The report of European working group on sarcopenia in older people says that the principle in using 2 criteria i.e, lower skeletal muscle mass and muscle performance is that strength of the muscle does not depend mainly on skeletal muscle mass and the relationship between strength and mass is not a linear. The EWGSOP report also divided sarcopenia into three stage that reflects the severity of the condition.

1. Presarcopenia stage	characterised by low muscle mass without impact on muscle strength or physical performance
2. Sarcopenia stage	characterised by low muscle mass and low muscle strength or low physical performance
3. Severe sarcopenia	characterised by low muscle mass, low muscle strength and low physical performance

EWGSOP conceptual stages of sarcopenia

Stage	Muscle mass	Muscle strength		Performance
Presarcopenia	↓			
Sarcopenia	↓	↓	Or	↓
Severe sarcopenia	↓	↓		↓

The European society of parenteral and entral nutrition special interest groups effort defines sarcopenia as “sarcopenia is a condition characterized by loss of muscle mass and muscle strength. Although sarcopenia is primarily a disease of the older individual , its development may be associated with condition that are not exclusively seen in older person, like disuse, malnutrition and cachexia. Like osteopenia, it can also be seen in younger patients such as those with inflammatory disease. The loss of muscle mass and muscle strength caused by such condition is usually functionally less relevant in younger individual, as their muscle mass and muscle strength is higher before it is affected by these condition”

The international working group on sarcopenia consensus wordings are “sarcopenia is defined as the age associated loss of skeletal muscle mass and function. The cause of sarcopenia was multi factorial and can include disuse, insulin resistance and nutritional deficiency. While cachexia may be a component of sarcopenia, the two condition are not the same”

The society of Sarcopenia, Cachexia and wasting disorders defines "sarcopenia, that is reduced muscle mass with limited mobility, should be considered an important clinical entity and defined as a condition with muscle loss and reduced gait speed".

PREVALENCE OF SARCOPENIA:

The occurence of sarcopenia showed in various research study varies considerably. This shows the difference in the elderly population studied, different method used to measure muscle mass and size and difference in the normal study group that were used to derive the sarcopenia threshold. Within the existing literature, the prevalence of sarcopenia in 60 – 70 years old is in the order of 5% to 13% [28]. These prevalence estimates increase to 11% to 50% for the population aged 8o years or older. Some reference estimated approximately 15% of people aged 60 to 69 years and approximately 40% of people older than 80 years [9]. The world health organization(WHO) suggest that there were 600 million people aged 60 or older in the year 2000 and that the number will increase to 1.2 billion by the year 2025 [29]. WHO count suggest that sarcopenia affects more than 50 million population today and it will affect more than 200 million population over next 40 years.

PATHOPHYSIOLOGY:

Sarcopenia has been contributed by many factors that include normal ageing changes, malnutrition, lack of physical activity, bed rest, chronic illness, smoking and certain drug therapy [30–32].

The pathogenesis involved are protein synthesis, protein degradation, integrity of the neurological and muscular system and contents fat in muscles.

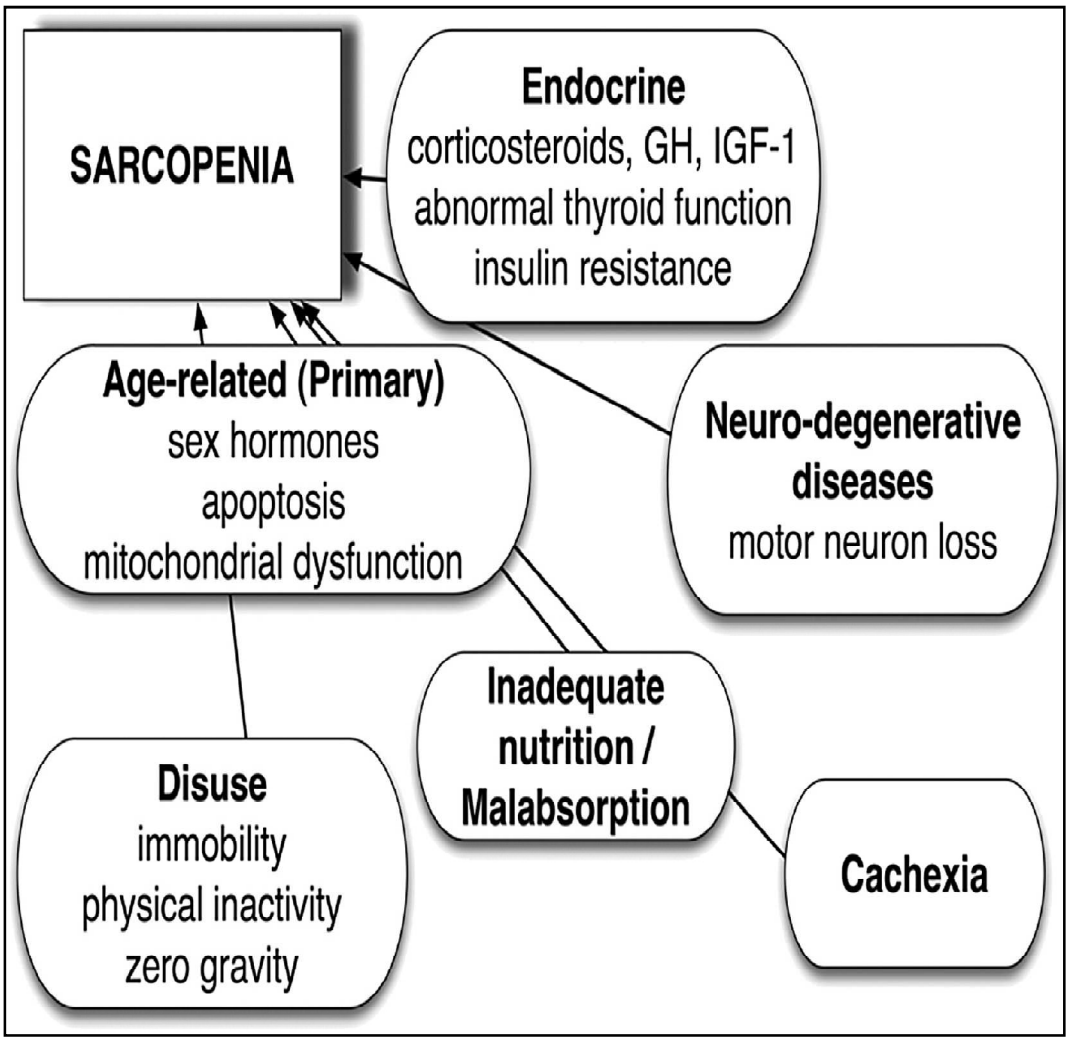


FIG.3 PATHOPHYSIOLOGY OF SARCOPENIA

The progression and development of sarcopenia was contributed by many factors. These risk factors are categorized as

- Constitutional factors
- Ageing process
- Life habits – decrease protein, disuse, tobacco and alcohol.
- Changes in living conditions – prolonged bed rest and immobility.
- Chronic health conditions.

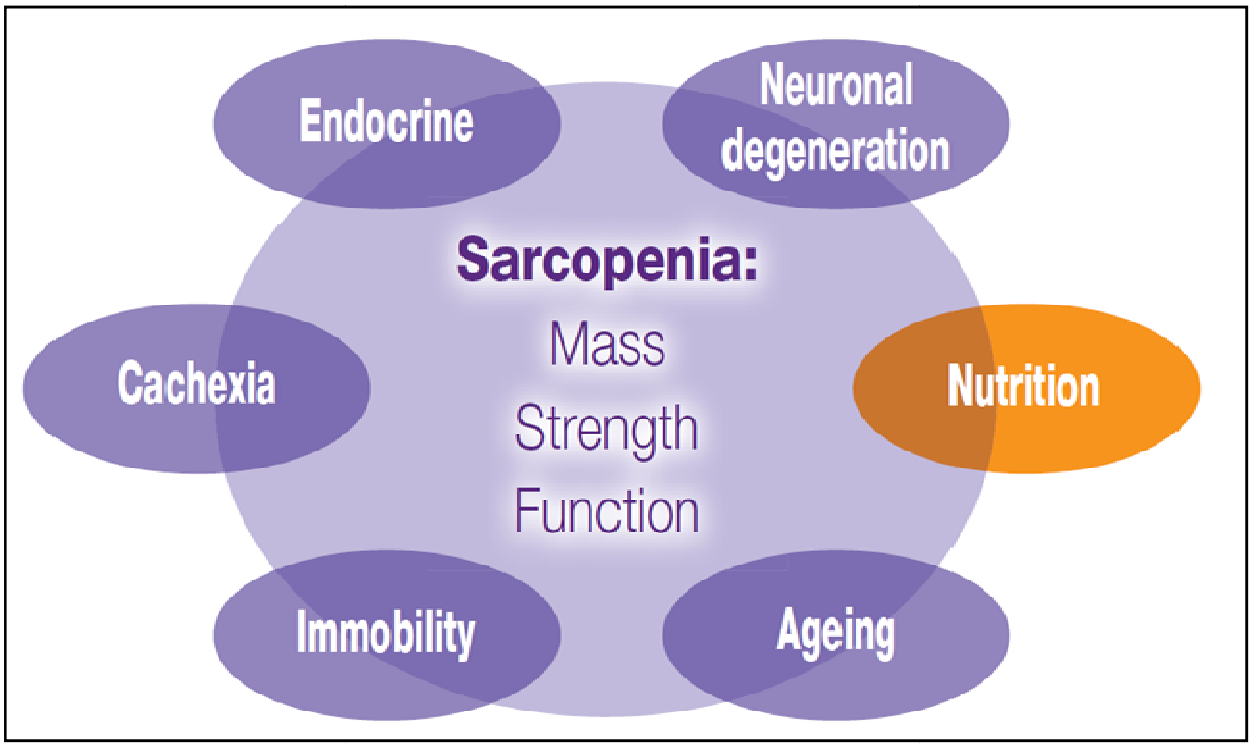


FIG.4 CONTRIBUTING FACTOR FORSARCOPENIA

Many mechanism attribute to the process of Sarcopenia the contribution varies for every individual.

The mechanism of sarcopenia is a complex process due to the ageing changes in musculoskeletal system in its structure as well as performance [33].The contributing and risk factors include sedentary life style, smoking, malnutrition, inadequate intake, obesity, ageing hormonal changes, neural changes and metabolic factor. Genetic factors also play a role insarcopenia.

The etiology of sarcopenia have beendivided into “intrinsic” and “extrinsic” factors. Reduced in anabolic hormonal level (DHEA, Estrogens, Growth hormones, IGF-1), increase in apoptosis of muscle fibers, increases of inflammatory cytokines (e.g. TNF-α, IL-6), free radical injury, mitochondrial dysfunction in muscles and a decrease in the number of α-moto neurons are the intrinsic factors. Decreased intake of protein and essential amino acids, low level of vitamin D, acute and chronic illness and sedentary life style are the extrinsic actors leads to sarcopenia.

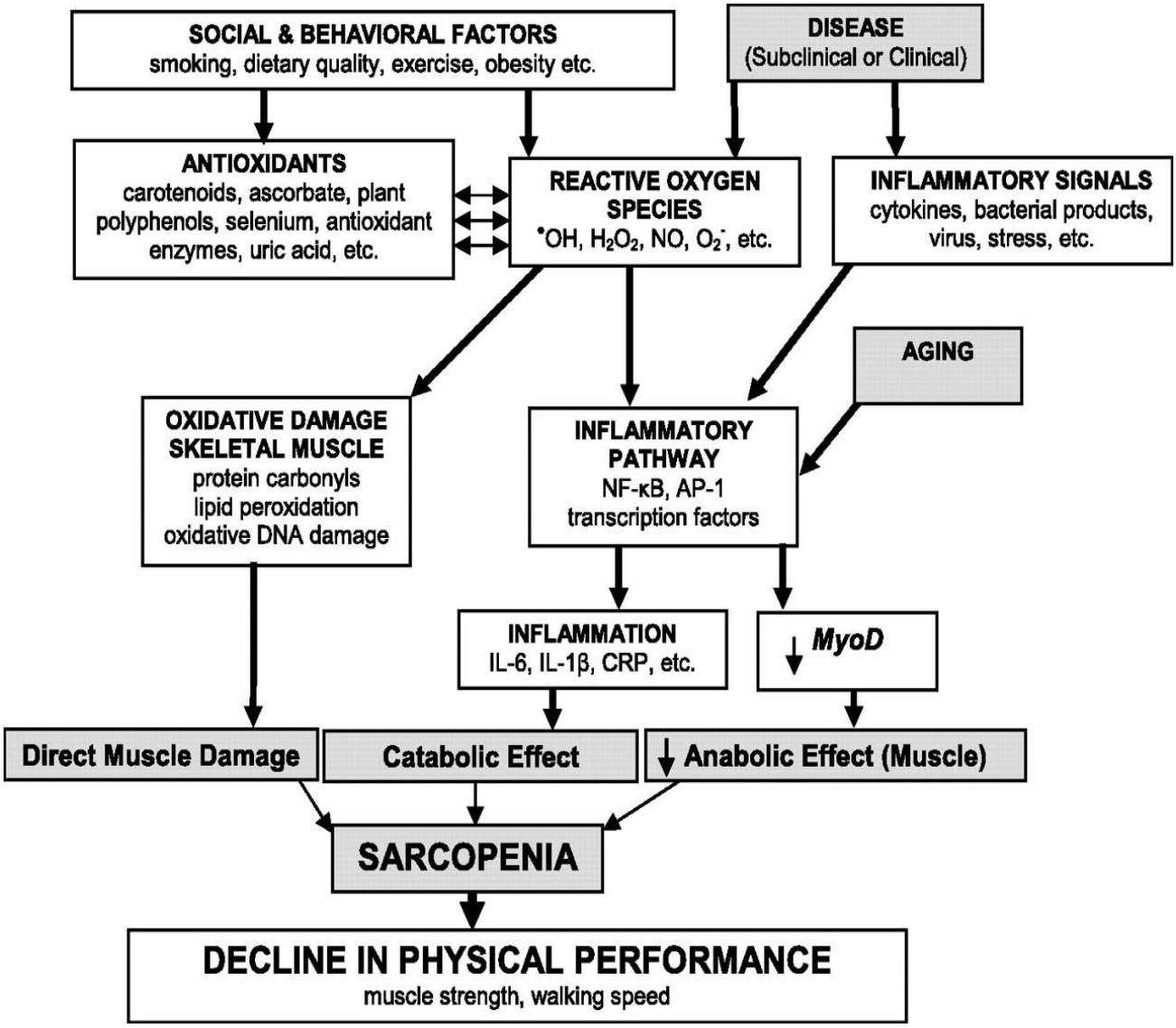


FIG.5 PHYSIOLOGY OF SARCOPENIA

The development of sarcopeniai.e, deline in muscle mass and strength due to mismatch between loss and renewal of muscle protein. The imbalance between atrophy or apoptosis and hypertrophy and satellite cell production.

SARCOPENIC OBESITY:

In conditions such as malignancy, rheumatoid arthritis and ageing, lean body mass is lost while fat mass may be preserved or even increased [34]. This state is called sarcopenic obesity, and thus the relationship between age-related reduction of muscle mass and strength is often independent of body mass. It had long been thought that age-related loss of weight, along with loss of muscle mass, was largely responsible for muscle weakness in older people [35]. However, it is now clear that changes in muscle composition are also important, e.g.'marbling', or fat infiltration into muscle, lowers muscle quality and work performance [36]. While weight changes vary widely between individuals, certain patterns of age-related change in body composition have been observed. In ageing men, the percentage of fat mass increases initially, then levels off or decreases. Such change has been attributed to an accelerated decrease in lean mass, along with an initial increase and a later decrease in fat mass [37]. Women show a generally similar pattern [37]. Intramuscular and visceral fat increase with ageing while subcutaneous fat declines [38,39].

ASSESMENT TECHNIQUE FOR SARCOPENIA:

MUSCLE MASS:

A variety of method has been used for the assessment of skeletal muscle mass [40].The elements that determine the assessment method as a suitable one for clinical and research purpose are

- Cost
- Acceptable
- Reproducible
- Ease of use

Body imaging techniques.

The estimation of skeletal muscle mass and lean body mass done by three imaging technique

1) Computed tomography (CT scan)

2) Magnetic resonance imaging (MRI) and

3) Dual energy X-ray absorptiometry (DXA).

Computed tomography and Magnetic resonance imaging can differentiate fat tissue other soft tissues hence consider as gold standard technique for the purpose of research. Because of the cost, ease to access and radiation risk these imaging technique is not used as a routine method in research and clinical study [41].dual energy X ray absorptiometry is the alternative method both for research and clinical practice use to differentiate fat mass, bone and lean body mass.

Bioimpedance analysis.

Bioimpedance analysis (BIA) evaluates fat volume, skeletal muscle mass and lean body mass. The advantage of technique over the other are inexpensive, easy to assess, reproducibility and suited or both the ambulatory and bedridden elderly population.

In standard conditions, the acceptability of the technique was studied for more than ten years[35], and bio impedance analysis results under standard conditions has been correlated well with Magnetic Resonance Imaging predictions [43]. Prediction equations have been validated for multiethnic adults and reference values established for adult white men and women, including older subjects [44–46].Thus, Bio Impedance Analysis is a good alternative to Dual energy xrayabsorpsiometry.

Total or partial body potassium per fat-free soft tissue.

As skeletal muscle contains more than fifty percent of the total body potassium (TBK) pool, thus total potassium pool is a best methodfor skeletal muscle estimation. Many recent studies have proposed the use of partial body potassium (PBK) of the arm as a better alternative [47]. PBK of the arm is safe and inexpensive. Though the total body potassium pool is the best method for skeletal mass estimation, this method is not used in clinical practice.

Anthropometric measures.

Mid upper arm circumference and skin fold thickness can used for estimation of skeletal muscle mass. Measurement of Calf circumference correlates positively with muscle mass. The measure of calf circumference less than 31 cm has been linked with the occurrence of disability [48]. As the calf circumference, mid upper arm circumference and skin fold thickness are not reliable for the measurement of sarcopenia they are not used in routine clinical practice.

MUSCLE STRENGTH:

The techniques validated for the estimation of muscle strength includes

1. Hand grip strength
2. Knee flexion or extension
3. Peak expiratory flow

The relevant method for the assessment of gait and physical function is the use of lower limb than upper limb. Although lower limbs are more relevant the widely used method has been hand grip strength. The cost, availability and ease of useare the elements that determine the assessment method as a suitable one for clinical and research purpose.

Handgrip strength.

Isometric hand grip strength is associated with

- Lower extremity muscle power,
- Knee extension torque and
- Calf cross-sectional muscle area [49].

The mobility problems and its clinical outcomes is better assessed by the hand grip strength than the skeletal muscle mass. Handgrip strength is a best simple method for the estimation of muscle strength, and also it correlates with leg strength.

Knee flexion/extension.

Muscle strength represents the amount of force generated and muscle power represents the rate of work. In healthy elderly the muscle power i.e, the rate of work done by the muscle lost faster than the muscle strength. Both muscle power and muscle strength are vital for the performance, but muscle power is a best determinant of certain functional activities [50 - 52]. Both the isometric muscle strength and isokinetic muscle strength measurementscan made using modern commercial isokinetic dynamometers. Knee flexion techniques need special equipment so it is not feasible in clinical practice only suitable for research studies.

Peak expiratory flow.

Peak expiratory flow (PEF) is determined by the strength of respiratory muscles in people without lung disorders. PEF is a cheap, simple and widely accessible technique which has prognostic value [53] even though it is not an recommended single measure.

PHYSICAL PERFORMANCE:

The various test used for the assessment of muscle performance includes,

- Short Physical Performance Battery (SPPB),
- Usual gait speed,
- 6-min walk test and
- The stair climb power test.

Short Physical Performance Battery.

The Short Physical Performance Battery includes variety of test evaluation of balance, gait speed, muscle strength and endurance. The SPPB includes the following test 1) ability to stand side by side with feet together 2) ability to stand in semi tandem posture 3) ability to stand in tandem position 4) eight feet walking test 5) chair stand test for 5 times. The Short Physical Performance Battery, a comprehensive measure of physical performance used both in the clinical practice and research.

Gait speed:

Gait speed is a reliable method for the assessment of physical performance. Even though gait speed is part of the Short Physical Performance Battery it can also be used as a single parameter for research and clinical practice.

Timed get-up-and-go test:

Intimed get-up-and-go test (TGUG) the subjects was required to sit in a distance of 3meter and then ask to get up walk for the distance, turn around come back and sit in the chair. The time taken for this series of task was calculated. It assess both balance and gait. Balance function is assessed and scored in a five point scale. The Timed get up and go test, used in comprehensive geriatric assessment scale, and used for performance measurement.

Stair climb power test.

The leg muscle power, strength and impairment can be measured clinically by the stair climb power test (SCPT). The Stair climb power test isuseful in some clinical research.

Measurements of muscle mass, strength, and function in research and practice[a]

Variable	Research	Clinical practice
Muscle mass	Computed tomography (CT)	BIA
	Magnetic resonance imaging (MRI)	DXA
	Dual energy X-ray absorptiometry (DXA)	Anthropometry
	Bioimpedance analysis (BIA)	
	Total or partial body potassium per fat-free soft tissue	
Muscle strength	Handgrip strength	Handgrip strength
	Knee flexion/extension	
	Peak expiratory flow	
Physical performance	Short Physical Performance Battery (SPPB)	SPPB
	Usual gait speed	Usual gait speed
	Timed get-up-and-go test	Get-up-and-go test
	Stair climb power test	

CUTOFF POINTS:

Cut-off points depend upon the measurement technique chosen and on the availability of reference studies. EWGSOP recommends use of normative (healthy young adult) rather than other predictive reference populations, with cut-off points at two standard deviations below the mean reference value.

MUSCLE MASS:

Baumgartner et al. calculated the Skeletal muscle index by summing the muscle mass of all the four limbs using Dual energy x ray absorpsiometry as appendicular skeletal mass which is divided by height2. Skeletal Muscle Index = ASM/h2 (kg/m2). A Skeletal Muscle Index 2 standard deviations below the mean Skeletal Muscle Index of healthy young male and female reference groups was defined as the gender specific cut off point for sarcopenia[66]. This method depends upon a measurement of appendicular skeletal muscle mass by DXA or estimation by BIA. SMI can also be calculated as skeletal muscle mass/height2. NHANES study used absolute skeletal muscle mass for the calculation of SMI. EWGSOP recommends both or the evaluation of muscle mass.

BIA	SMI using BIA predicted skeletal muscle mass (SM) equation ($SM/height^2$) Men: 8.87 kg/m^2 Women: 6.42 kg/m^2	Based on 2 SD below mean of young adults in study group (n = 200)
	SMI using absolute muscle mass, not appendicular muscle mass (absolute muscle mass/$height^2$) Men: Severe sarcopenia ≤8.50 kg/m^2 Moderate sarcopenia 8.51–10.75 kg/m^2 Normal muscle ≥10.76 kg/m^2 Women: Severe sarcopenia ≤5.75 kg/m^2 Moderate sarcopenia 5.76–6.75 kg/m^2 Normal muscle ≥6.76 kg/m^2	Based on statistical analysis of NHANES III data on older (≥60 years) men and women

Muscle strength measured based on hand grip strength using hand held Dynamometer based on BMI can be a surrogate marker for muscle strength. Hand Dynamometer is a very simplest method and a acceptable, reproducible method to measure muscle strength.

Handgrip strength	Men: <30 kg	Based on statistical analysis of study group (n = 1,030)
	Women: <20 kg	
	Men:	Based on quartiles of study group (n = 5,317)
	BMI ≤ 24 ≤ 29 kg	
	BMI 24.1–26 ≤ 30 kg	
	BMI 26.1–28 ≤ 30 kg	
	BMI > 28 ≤ 32 kg	
	Women:	
	BMI ≤ 23 ≤ 17 kg	
	BMI 23.1–26 ≤ 17.3 kg	
	BMI 26.1–29 ≤ 18 kg	
	BMI > 29 ≤ 21 kg	

GAIT SPEED:

The EWGSOP has suggested algorithm based on gait speed measurement as the easiest and most reliable way to begin sarcopenia

GAIT SPEED:

Gait speed can be used for case finding or screening in practice. A cut-off point of more than 0.8 m/s identifies risk for sarcopenia [54]. EWGSOP recommends gait speed as the initial test for the screening of sarcopenia.

EFFECTS OF SARCOPENIA:

In the year 2000, 35 million were in the age group of 65 years and it was increased to forty million in the year 2010, a increase of fifteen percent was noted. This population may further increase to fifty five million by the year 2020, a raise of 36% for that decade. By the year 2030, there will be about 72.1 million elderly population, about double compared to the population at 2008[55].

In 2000, 1.5 million elderly population were hospitalized, and 33% which represents one third of the institutionalized population were admitted to longterm health care facilities due to their inability in performing ADL[56]**.**Sarcopenia, the decline in muscle mass and muscle performance is thought to affect thirty percent of population over the age of 60 years and more than fifty percent of population over 80 years of age [57].

Recent analysis shows that nearly 45% of elderly population in United states is affected by the sarcopenia. This corresponds to 18 million population in the year 2010 it was still continue to rise [58].

In the U.S. population around 20% of elderly population were functionally disabled [59].The risk of disability is 1.5 to 4.6 times higher in elderly with sarcopenia compared to elderly with normal muscle mass and strength [60].

The fall risk will raise in the population associated with sarcopenia. Nearly half of accidental deaths in the population of age above 65 are related to falls. Though nutritional supplement, diet and exercise can reduce the rate of muscle mass and strength loss, even healthy and ambulant elderly will experience decline in muscle function [61].

COST OF SARCOPENIA:

The estimated direct healthcare cost attributable to sarcopenia in the United States in 2000 was $18.5 billion ($10.8 billion in men, $7.7 billion in women), which represented about 1.5% of total healthcare expenditures for that year. [60]

Healthcare expenditures due to sarcopenia cost roughly $900 per person per year [62]. People with chronic illnesses and activity limitations caused by conditions like sarcopenia have more physician visits and fill more prescriptions than those individuals with no activity limitations, all of which presents a greater burden on our health care system. These individuals also have more health visits [63].

The United States spends more than $26 billion annually on additional health care costs for people over 65 who lose their ability to live independently over the course of a single year [64]. A 10% reduction in the sarcopenic population would save $1.1 billion. Though sarcopenia

contributes to numerous other health problems and accounts for a similar percentage of healthcare costs as osteoporosis, no public health campaigns are directly aimed at reducing the prevalence of sarcopenia.[60]

Thusby maintaining the functional independence and physical function in elderly population will preserve the good quality of life and also save health care cost of our country too.

ASSESMENT OF SARCOPENIA:

EWGSOP recommended an algorithm for screening of sarcopenia. It can to be done all person aged above 65 years and also in adults with risk of sarcopenia.

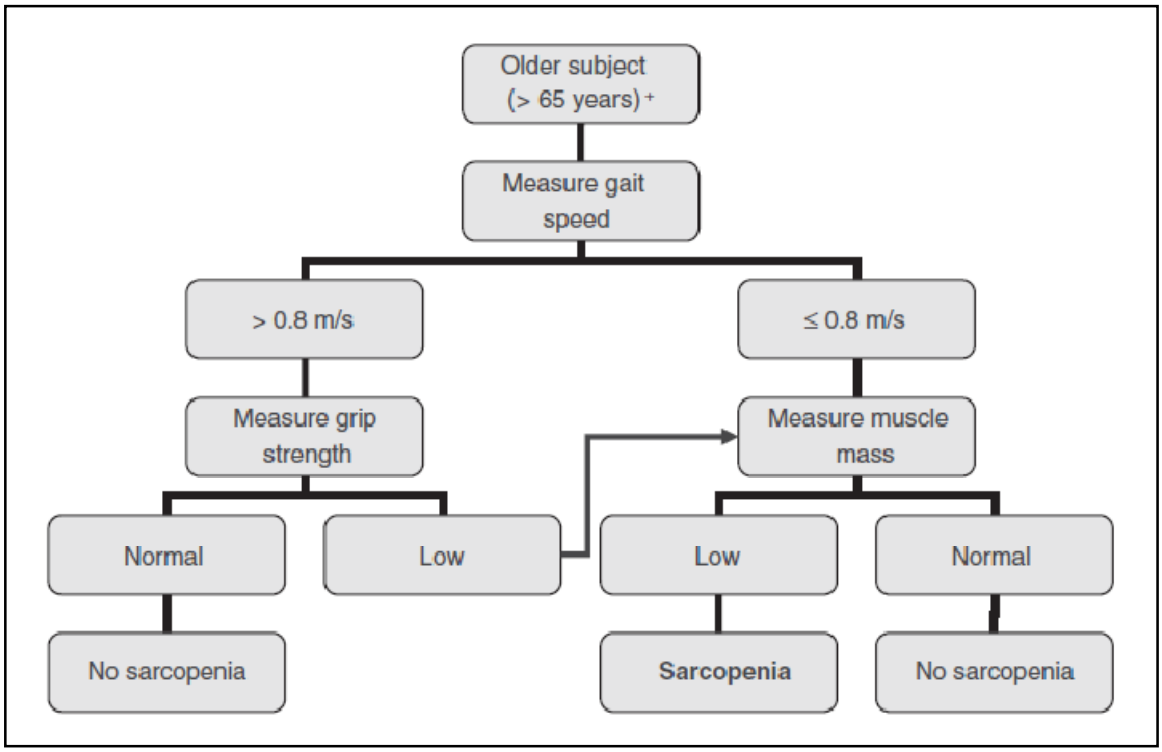


FIG.6 ALGORITHM FOR ASSESMENT OF SARCOPENIA

VITAMIN D

Lipid soluble vitamin apolar hydrophobic molecule which are all isoprene derivatives, they generally cannot be synthesized by the body in adequate amount and therefore must be supplied in the diet. They efficiently absorbed with the fat dietand once absorbed bound to lipoprotein or specific binding protein and transported to the specific organ. The important fat soluble vitamins are vitamin A, B, C, D, E and K. Vitamin D is a steroid pro hormone. It is synthesised from steroid that occurs in animal, plant and yeast which undergo many metabolic changes inside the human body and give rise to a hormone called as calcitriol which play a major role in calcium and other metabolic action.

Vitamin D, is lipid soluble vitamin and an important nutrient in our body. 1,25dihydroxycholecalciferol [1,25$(OH)_2$D] is the major steroid hormone in our body. It is involved in mineral ion homeostasis regulation primarily. Vitamin D and its metabolites are mainly hormones and hormone precursors rather than to be called as vitamins, as 1,25$(OH)_2$D is synthesized endogenously. UV rays acts on the skin and causes 7-dehydrocholesterol to cleave by a photochemical reaction and results in Vitamin D. If melanin content is more in skin the production of Vitamin D by skin is reduced. Similarly sunscreens having high SPF even more than 8 also reduces the production by impairing the penetration of UV rays into the skin. The

increased use of sunscreens all over the world for cosmetic purpose and a reduced exposure to sunrays over the last several decades led to an increased dependence on dietary source of vitamin D.

CHEMISTRY AND STRUCTURE OF VITAMIN D:

Vitamin D is a secosteroid molecule it is not classified as a essential nutrient as it is produced in abundant by the skin on exposure to sunlight. It function as a hormone in the body to play a key role in the overall metabolism of bony skeleton.

Its structure is similar to that of cholesterol. It differs from cholesterol by presence of double bonds between c7 c8 and c 19 and an open ring structure. The two forms of vitamin D utilised in the human body D2 and D3 begin with four intact rings. The body half life of vitamin D3 molecule is 62 days. This estimate is based on radioactively labelled vitamin D3. During synthesis Ultraviolet B (UV-B 290-310nm) breaks the B ring of both compounds by cleaving the bond between C-9 and C-10 of B ring followed which a formation of double bond between C-10 and C-10.

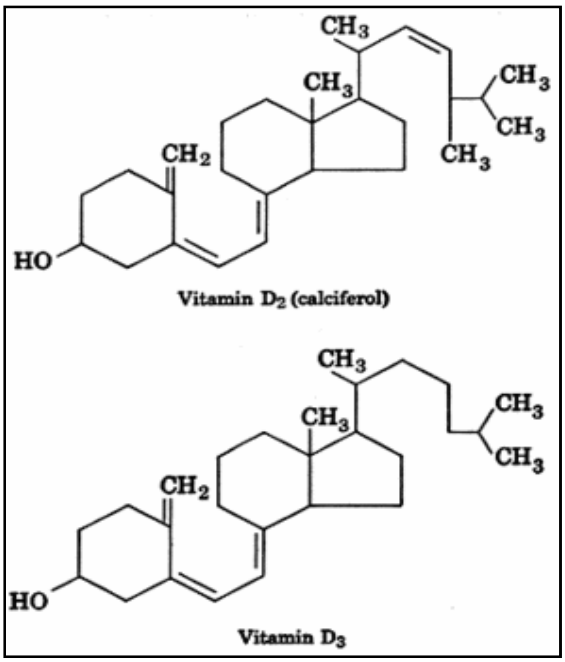


FIG.7 STRUCTURE OF VITAMIN D

FOOD SOURCE:

Food fortification is a common sources of vitamin D [65,66] Somecereals may be fortified with vitamin D. There are only a few commonly consumed foods that are good sources of vitamin D [65]. Cod liver oil, fish, egg yolk, cereals, yogurts, fortified orange juice, mushroom, soya, liver and beef are some of the major food sources of vitamin D. The plant sources of contains vitamin D2 and the animal source contain Vitamin D3. Cod liver oil contains the maximum amount about 1360 IU per serving. Most of the non vegetarian food contains the vitamin D.

Food	IUs per serving*	Percent DV**
Cod liver oil, 1 tablespoon	1,360	340
Salmon (sockeye), cooked, 3 ounces	794	199
Mushrooms that have been exposed to ultraviolet light to increase vitamin D, 3 ounces (not yet commonly available)	400	100
Mackerel, cooked, 3 ounces	388	97
Tuna fish, canned in water, drained, 3 ounces	154	39
Milk, nonfat, reduced fat, and whole, vitamin D-fortified, 1 cup	115-124	29-31
Orange juice fortified with vitamin D, 1 cup (check product labels, as amount of added vitamin D varies)	100	25
Yogurt, fortified with 20% of the DV for vitamin D, 6 ounces (more heavily fortified yogurts provide more of the DV)	80	20
Margarine, fortified, 1 tablespoon	60	15
Sardines, canned in oil, drained, 2 sardines	46	12
Liver, beef, cooked, 3.5 ounces	46	12
Ready-to-eat cereal, fortified with 10% of the DV for vitamin D, 0.75-1 cup (more heavily fortified cereals might provide more of the DV)	40	10
Egg, 1 whole (vitamin D is found in yolk)	25	6
Cheese, Swiss, 1 ounce	6	2

FIG.8 SOURCES OF VITAMIN D

SYNTHESIS:

Vitamin D either synthesized from the skin or absorbed from intestine enters into the circulation, bound to vitamin D–binding protein (DBP). DBP is an α-globulin synthesized in the liver. Ergosterol occurs in plants and invertebrate animals and 7 dehydrocholesterol occurs in vertebrate animal. Ergosterol differs from 7 dehydro cholesterol only in its side chain which is unsaturated and contains an extra methyl group. Vitamin D is generated from the pro vitamin, Dehydrocholesterol by the action of sunlight. Ultraviolet B

(UV-B 290-310nm) breaks the B ring of both compounds by cleaving the bond between C-9 and C-10 of B ring followed which a formation of double bond between C-10 and C-10. In plants ergocalciferol (vitamin D2) is formed and in animals cholecalciferol (vitamin D3) is formed from sunlight.

Dietary vitamin D2 or D3 observed from the gut in micelles and transported into the lymphatics after incorporation into chylomicrons and then enters into systemic circulation where it is bound to vitamin D binding protein (glycoprotein).vitamin D 3 formed from 7 dehydro cholesterol also enters into the blood and it is also bound to vitamin D binding protein. This complex of Vitamin D is hydroxylated in the liver to 25(OH)D by cytochrome P450–like enzymes in the mitochondria and microsomes. This hydroxylase step in liver is not tightly regulated. 25(OH)D is the major circulating form.

Vitamin D is stored as 25(OH)D. 88% of 25(OH)D is bound to the vitamin D–binding protein and circulates in the body. 0.03% is in free form and remaining 25(OH)D circulates in the body along with albumin. The half-life of 25(OH)D is approximately 2 -3weeks. It is shortened dramatically if Vitamin D–binding protein levels are decreased, as in nephrotic syndrome where there is urinary loss of albumin. Second hydroxylation takes place in the proximal tubular cells by the enzyme 1, 25, hydroxylase which converts 25(OH) D to 1, 25, $(OH)_2$ D and enters into circulation which has a half life

of 4 hours and becomes active on binding to VDR. Thus calcitriol, an active but unstable 1, 25 dihydroxy Vitamin D [1, 25(OH)2-D] cholecalciferol (Calcitriol) synthesised.

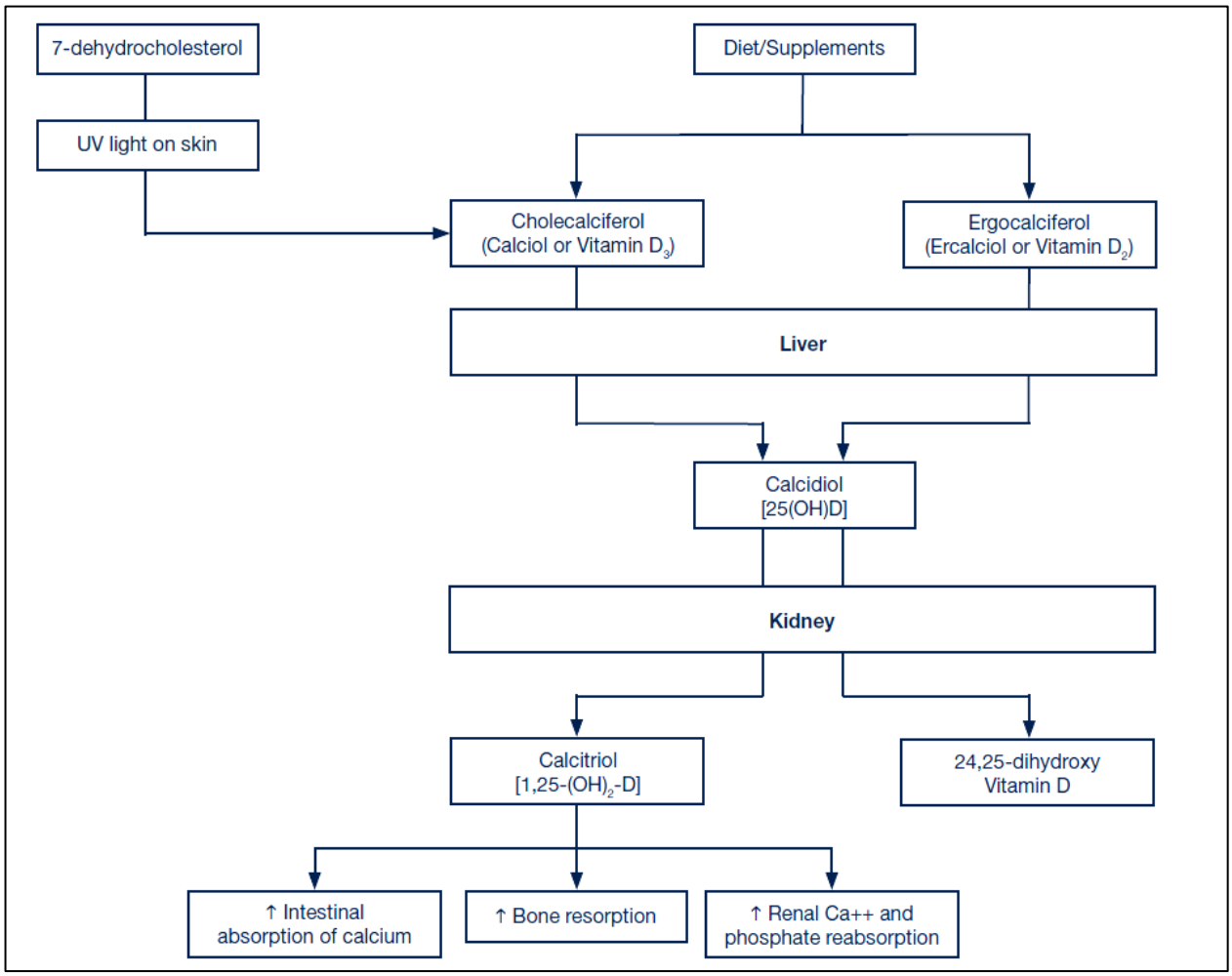


FIG.9 SYNTHESIS AND FUNCTION OF VITAMIN D

Thus physiological action of vitamin D on bone and other organ system occurs through calcitriol which is the active form of vitamin D. The storage form was 25 Hydroxycholecalciferol.

SYNTHESIS OF VITAMIN D

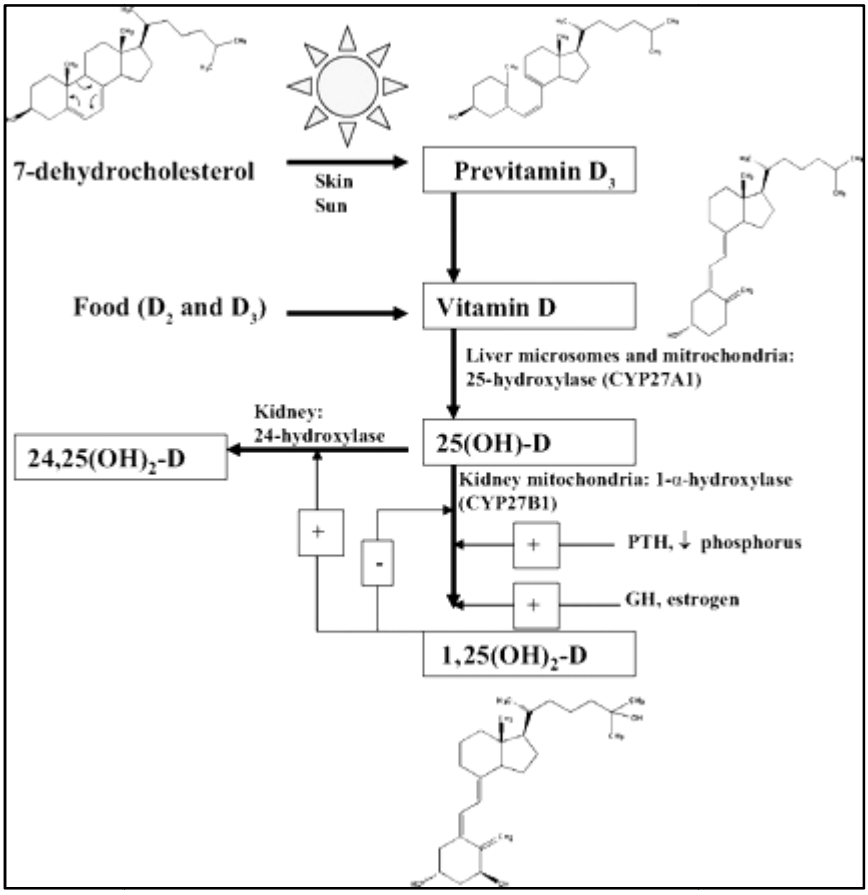
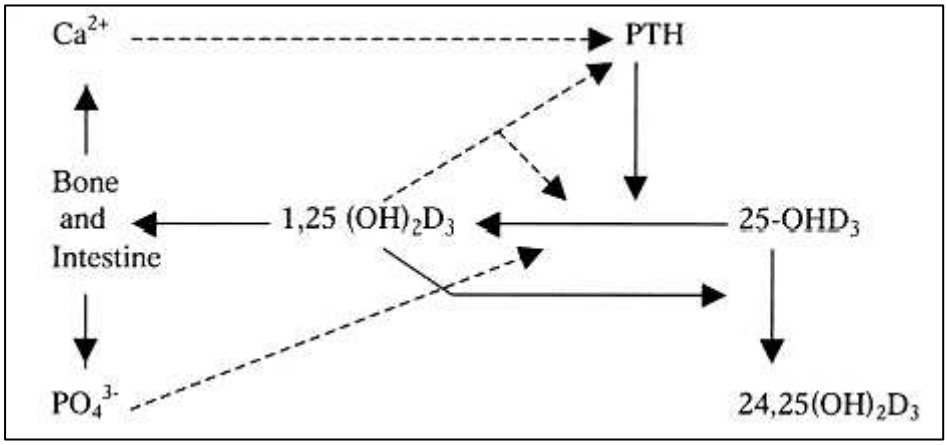


FIG 10 SYNTHESIS OF VITAMIN D

REGULATION OF VITAMIN D SYNTHESIS:

The ingested vitamin D which is converted into 25(OH) D3 in the liver is regulated. The level of 25(OH) D3 reflects the quantity of Vitamin D generated in the skin or in the dietary intake. In contrast, the control of synthesis of metabolically active vitamin D is rom 1, alpha hydroxylase enzyme in the kidney. 1-alpha-hydroxylase is induced by the parathyroid hormone. Low phosphate level is an inducer of 1, alpha hydroxylase enzyme

[67].Parathyroid Hormone by acting on a kidney tubular membrane receptor reduces the phosphate (as well as HCO3-) reabsorption from PCT. Parathyroid hormone also increase the ca+ uptake from DCT and induces the conversion of 25(OH) Vitamin D3 to 1,25 (OH) Vitamin D3 and thus increases the calcium absorption from G.I tract.



FORMS OF VITAMIN D:

Although 5 forms of vitamin D are known (D1 – D5) D2 and D3 are the most studied form. The two major forms are vitamin D2 or ergocalciferal and vitamin D3 or cholecalciferal. Vitamin D without subscript refers to either D2 or D3 or both. These are knowm collectively as calciferol. Many research made on the significant function of vitamin D5. Some recent study shows that it has aanti tumour effect.D2 synthesised from plant source and D3 from the animal source. The table illustrate the forms of vitamin D

FORMS OF VITAMIN D

FORMS	CHEMICAL COMPOSITION	SIGNIFICANCE
D1	Combination of ergocalciferol and lumisterol	
D2	Ergocalciferol: made of ergosterol or provitamin d2	Made by invertebrates, fungus and plants in response to ultraviolet radiation.
D3	Cholecalciferol: made from 7-dehydrocholesterol or pre vitamin d3	Made in skin in response to ultraviolet b radiation after reacting with 7-dehydrocholesterol
D4	Dihydroergocalciferol: vitamin d2 without 22,23 double bond	Ineffective form of vitamin d
D5	Sitocalciferol: made from 7-dehtdrositosterol	May have anti tumour properties

PHYSIOLOGICAL ACTIONS OF VITAMIN D:

Intestines:

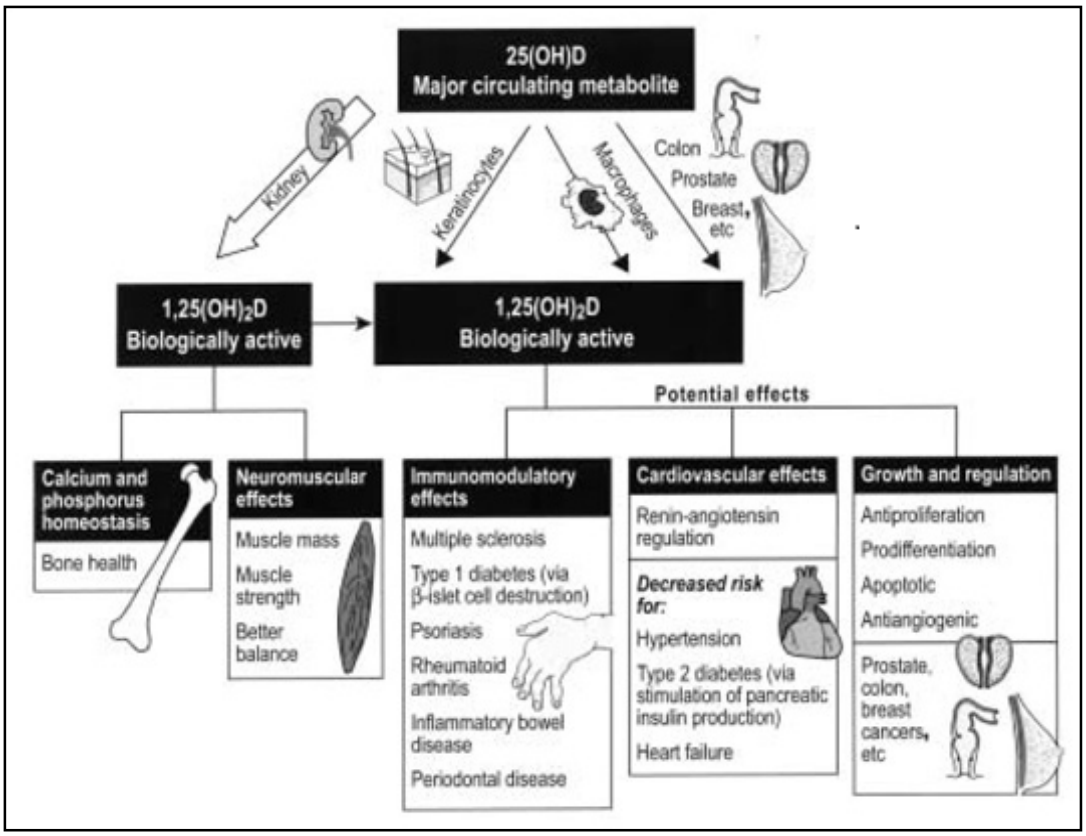
In the intestine calcitriol, active form of vitamin D stimulates the calcium and phosphate absorption and hence increases the level of blood calcium and phosphorous [68].

Bones:

By inducing ca+ absorption, calcitriol helps for the formation and maintenance of healthy bone. Calcitriol also act in concern with a many of other minerals, vitamins, and hormones for the promotion of bone mineralization [69].

Other functions:

Research suggests that vitamin D may help to maintain a normal arterial resistance thereby maintain blood pressure and also a preventive role in myocardial infarction an important complication of hypertension [70,71]. Also it has a role in maintenance of healthy immune system and help regulate cell growth and differentiation. It turns out that vitamin D receptors are present in all cells in the body [72]. Vitamin D helps in regulation of muscle strength via various mechanism. Many studies proved that vitamin D has a role in glycemic control by decreasing the insulin resistance. Still many function of Vitamin D are still in research.



Vitamin D plays an important role in many normal body functions, including:

— Regulation of cell growth.

— Bone formation.

— Immune function.

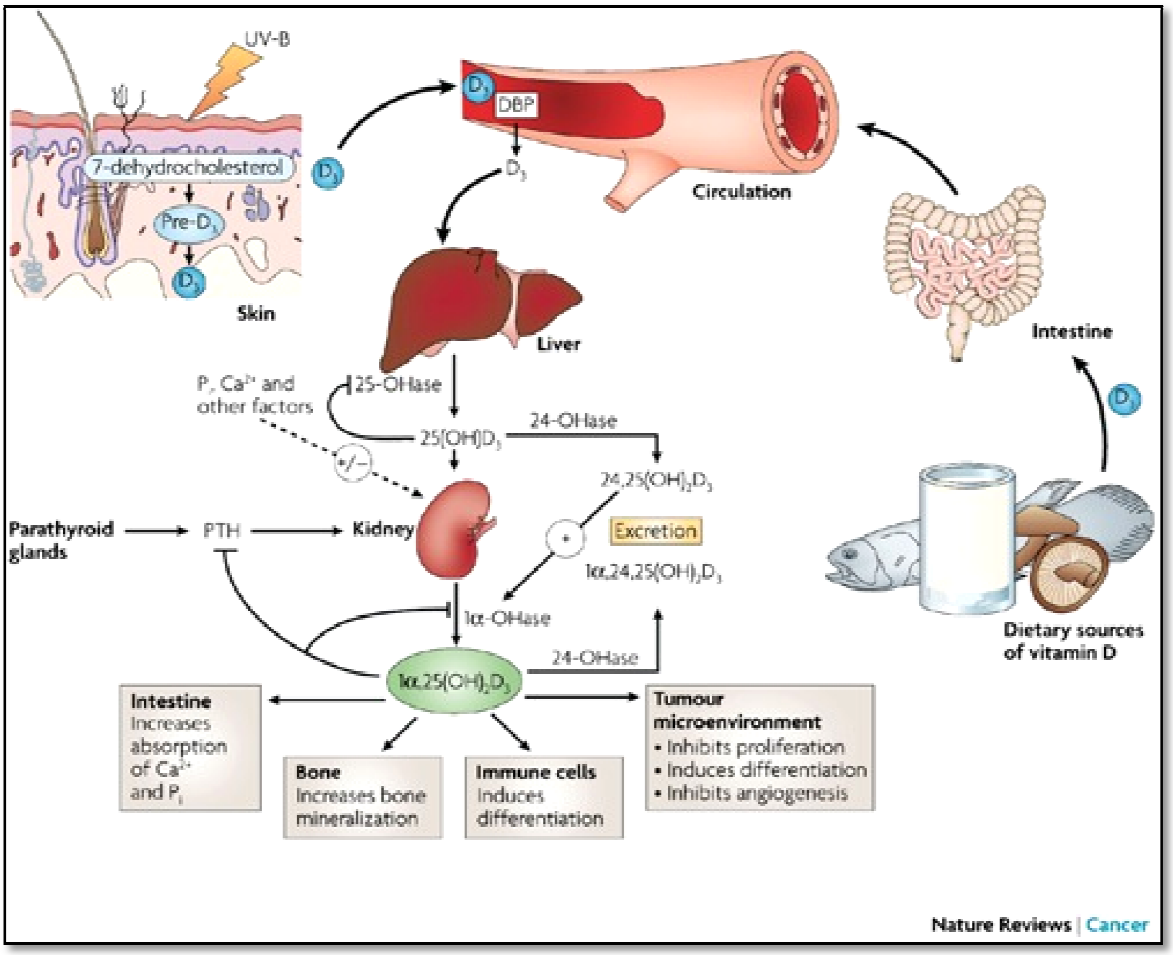
— Muscle strength.

— Hair growth.

— Fighting infections.

— Reducing the risk of autoimmune diseases.

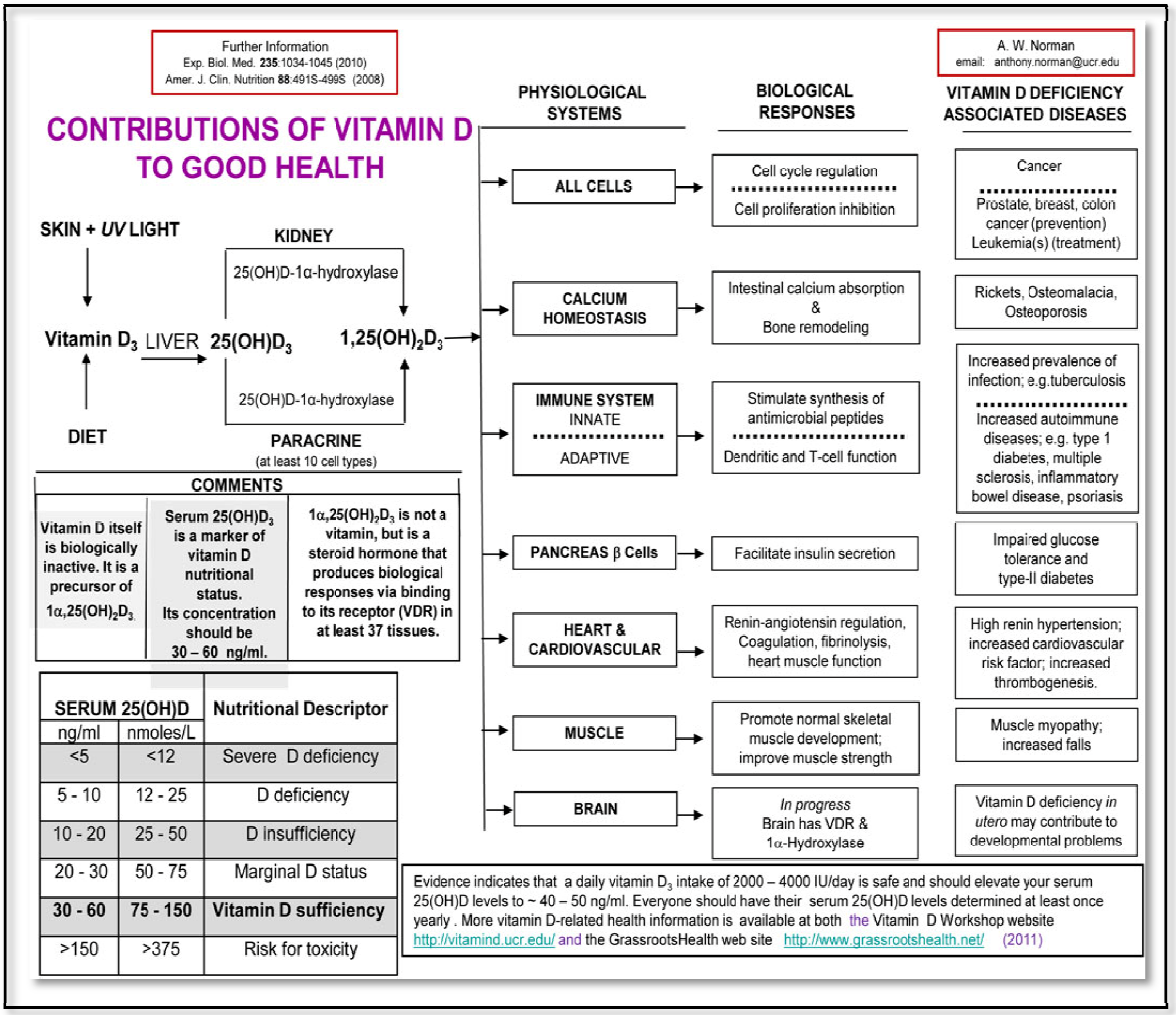
It is well accepted fact that Calcitriol is needed for the healthy bone as it play a major role in calcium and phosphorus metabolism. Many researches showed the expression of Vitamin D receptor in various tissue including muscle tissue. etal muscle. Severe vitamin D deficiency leads to rickets in childrens and osteomalacia in adults manifest as proximal muscle weakness. Histological finding shows atropic changes of skeletal muscle fibres especially in Type II muscle. There is contradictory evidence to show the contribution of Vitamin D for proximal muscle weakness. Some research showed a significant correlation and some doesn't show significant association. A recent study investigated the relation between quadriceps muscles trength and vitamin D levels demonstrates a significant association.



Apart from Vitamin D well understood role in bone and calcium metabolism, Vitamin D also plays a major role in multiple system that include endocrine, immunity, cardiovascular, neuropsychiatric functions, neuromuscular function and is also has a antioxidant property against free radical injury, as well as being an induction of cellular level differentiation, protecting cells from carcinogenesis [73]

PLEOTROPIC ACTIONS OF VITAMIN D:

Pleotropic concept on Vitamin D begins with two discoveries [74].The first is finding the expression of Vitamin DReceptors in many tissues other than bone tissue. Vitamin DReceptors are seen in many tissues that include cardiac tissue, gastro intestinal tissues, Hepatic tissue, Renal tissue, Lungs, Brain tissue, pancreas and many immunology cells. The second is the discovery of enzyme Cytochrome P27B1 which is capable of converting 25,hydroxy vitamin D into 1, 25 hydroxy vitamin D in many tissues throughout the body. These shows local autocrine and paracrine role of Calcitriol added to its function in endocrine [75]. Thus vitamin D in addition to calcium and bone homeostasis also has it role in many autocrine and paracrine function. Hence it is mentioned as pleotropic effect of vitamin D. In all cells it has a role in cell regulation and proliferation. In immune system it has a role in both the innate and adoptive immunity. In pancreas in facilitation of beta cells. It also plays some role in renin system. In muscles it plays role in muscle strength. These are some of the pleotropic effect of Vitamin D.



MECHANISM OF ACTION:

1,25$(OH)_2$D mediates its biologic effects by binding to VDR. VDR is a member of the nuclear receptor superfamily. This receptor belongs to the subfamily of receptors of thyroid hormone, the retinoid and PPAR, but only one isoform has been isolated. The VDR binds to target DNA resulting to recruit a series of co activators leading to modification of chromatin and

hence approximating the VDR to the basal transcriptional apparatus, ending in the induction of target gene expression. VDR is expressed in a wide range of cells and tissues. Thus it shows that vitamin D is not only a factor for bone and homeostasis also in many ass mentioned above. Many non ionic role of Vitamin D and Vitamin D Receptors is now a million dollar research subject and is used in prevention and progression of carcinomas, inflammatory diseases like Multiple sclerosis, Crohn's disease, Rheumatoid arthritis and Diabetes mellitus.

TEST FOR VITAMIN D:

Vitamin D status refers to the estimation of the vitamin stored in our body. The 25- hydroxyl vitamin D3 is widely acknowledged as the best indicator or determinant of the nutritional status of this fat soluble vitamin. The 25(OH) D directly relates to the body storage whereas 1,25(OH) Vitamin D although active metabolite correlates to the disorder involving vitamin D endocrine system. Vitamin D is measured in the serum in 25(OH)D form. 1, 25 $(OH)_2$ D is the active form and its levels are not reliable because it halflife is four hours within body and half hour to one hour outside the body. Whereas 25(OH)D present in serum is stable and can be measured for a day, if sample is kept in room temperature and 2-4^{o}c for 2-3 weeks and in -20^{o}c for a year.

25(OH)D levels are measured by variety of means like

- ✓ High Performance Liquid Chromatography (HPLC) and Mass spectrometry.
- ✓ Radio Immunoassays (RIA).
- ✓ Enzyme immunoassays (EIA).
- ✓ Competitive protein binding assays (CPBA).
- ✓ Automated chemiluminescence protein-binding assays (CLPBA).
- ✓ Chemiluminescent immunoassays (CLIA).

HPLC, RIA , EIA, CLIA are widely used currently. But there is no gold standard method available for measuring 25(OH) D levels.

NORMAL SERUM LEVEL AND DEFICIENCY:

Normal serum Vitamin D(25(OH)D) has wide range 30-100 ng/ ml. Vitamin D deficiency is considered to be present when serum 25(OH)D levels are <20ng/ml. It is said Vitamin D insufficiency if the levels are between 20-30ng/ml and called sufficient if the levels are >30ng/ml.It has been stated that most of the individuals have concentrations above 20 ng/ml and do not manifest any symptoms as this range is sufficient to maintain bone

health. But other metabolic functions of Vitamin D in active form are affected.

In our country none of the individuals should be Vitamin D deficient as we live in a country with plenty of sunshine . But various studies conducted have shown Vitamin D deficiency /insufficiency in about 50-90% implying Vitamin D deficiency is very common in India. Common in all age groups and in both sexes.Several studies conducted in North India also confirmed the prevalence to be about78-96%. Similar studies conducted in New Delhi by Goswami et al and Marwaha et al, also indicated a high prevalence of Vitamin D deficiency with a mean 25(OH)D 3 from 8.7±4.3 to 14±9.3ng/ml.

LEVELS OF VITAMIN D:

25(OH)D concentration	**ng/ml**	**nmol/L**
Vitamin D Deficiency	<10	<25
Insufficiency	10-30	25-75
Normal	30-100	75-250
Toxicosis	>100	>250

In India Vitamin D deficiency is a very common problem due to:

1. Low intake of Vitamin D and calcium due to food faddism and altering food habits [76].

2. The diet with more fibre content contains phytate and phosphate which will decrease the stored Vitamin D and also increase the requirement of calcium.

3. Genetic factors like increased 25,hydroxy D 24, hydroxylase which cause degradation of 25 hydroxy Vitamin D to inactive compound [77]

4. Hereditary factor of Vitamin D binding protein plays a role in the treatment response in the form of increased 25, hydroxycholecalciferol.[78]

5. With urbanisation, the time spent outdoors have decreased thereby reduces the adequate sun exposure. Ultra violet B, which plays a major role in Vitamin D synthesis has a short wavelength. Thus the maximum synthesis was between 10 A.M to 3P.M due to early scattering, the time where most of people were indoor.

6. Increased pollution also contributes to the restriction of ultraviolet rays to produce Vitamin D in the skin [79]

7. Cultural and traditional actors like Burqa and Pardah usage in muslims prevents the exposure to sunlight.

8. Ultra violet – B was more screened compare to Ultra violet A by the sunscreens. Sun screens with SPF 8 will reduce the synthesis by 95% and SPF 15 will reduce the synthesis by 98%. The usage of sunscreens become more prevalent nowadays.

9. Multiparity and unspaced pregnancy can lead to increased need that aggravate the Vitamin D deficiency both in mother and fetus.

10. Vitamin D deficiency is more prevalent in elderly people because they stay more in indoors and also have poor dietary intake and they are often have at least subclinical deficiency.

Vitamin D deficiency in elderly is thought to occur mainly due to restricted sunlight exposure, reduced dietary vitamin D intake, and decreased capacity of the skin to produce vitamin D.MacLaughlin and Holick et al examined the effects of ageing on the capacity of the skin to produce previtamin D3 in the skin by comparing young subjects (8 and 18 years old) with aged subjects (77 and 82 years old). They showed that ageing decreased the capacity less than half of young subjects , suggesting that elderly people are potentially at risk for vitamin D insufficiency/deficiency.

RECOMMENDED ALLOWANCES:

AGE	CHILDREN	MEN	WOMEN	PREGNANT
UPTO 13	200IU			
14 – 18		200IU	200IU	200IU
19 – 50		200IU	200IU	200IU
51 – 70		400IU	400IU	
OVER 70		600IU	600IU	

Recommended Daily Allowance are 400 IU/day for adults, 1000IU for babies upto 2yrs, 500-1000U for older children , and 400-800 IU for pregnant women. Requirements for vitamin D increase with ageing. 400 IU for those aged 51 to 70, and 600 IU for those over 70 years. These recommendations were established by determining the level of Vitamin D that was enough to prevent bone demineralization or rickets. The safe tolerable upper intake level for Vitamin D is 10,000 IU/day.52 Randomized trials using the currently recommended intakes of 400 IU of Vitamin D/day have shown no appreciable reduction in fracture risk after 50 yrs. In contrast, trials using 700‑800 IU Vitamin D/day found less fracture incidence.

SUPPLEMENT:

Supplements of Vitamin D are measured in both micrograms (µg or mcg) and International Units (IU).

1 microgram (µg or mcg) is equivalent to 40 IU of vitamin D2 or D3.

Vitamin D supplements is of 2 types:

1. Vitamin D2 (ergocalciferol)

2. Vitamin D3 (cholecalciferol).

Cholecalciferol supplement cause higher and more constant blood levels of 25, hydroxy Vitamin D and it is the supplement of choice compared to Vitamin D2 [80]

SARCOPENIA AND VITAMIN D:

In vitamin D deficiency, the decline in strength of the proximal muscle is one of the prominent feature clinically. Based the invention of Vitamin D receptor in the skeletal muscle and its associated decline with ageing many interventional studies made to demonstrate the therapeutic effect of Vitamin D. Many large scale studies demonstrate a low Vitamin D level with increased decline in muscle strength, performance, postural instability, walking speed and balance.

Many morphological changes in muscle was associated with Vitamin D deficiency. For example, patients with osteomalacia with muscle dysfunction associated with vitamin D deficiency show degenerative changes such as fat infiltration in muscle fibres, opaque fibers, ghostlike necrotic muscle fibers, regenerating and fibrosed muscle, enlarged interfibrillar spaces, fibrosis, glycogen granules, andatropy of the type II muscle fiber**.** As is the case with vitamin D-deficient patients, it is well known that elderly people show aberrant muscle morphology.

In older individuals, vitamin D deficiency leads to not only impaired bone mineralization but also muscular myopathy. Multiple crosssectional studies in older people have demonstrated an association between low levels of 25(OH)D and loss of muscle strength [82 – 84, 91], sarcopenia [82], balance [85,86], functional disability, limitations [83,86,87,91], and falls [85,88,89]. Kwon et al. reported that low serum 25(OH)D and albumin may be associated with a decrease in the objective physical performance of community-based older individuals in Japan [90]. Suzuki et al. reported that low serum 25(OH)D may be associated with physical performance and fall experience [92].

Vitamin D can exert its effects by genomic and non-genomic pathways. Both can be involved in muscle function. Besides the muscle cell (type II fibers), vitamin D could also influence neuromuscular action.

Evidence for the vitamin D receptor (VDR) in muscle cells and cell lines has been found by several investigators with different methods such as mRNA, calcium-binding protein, and VDR antibodies [93]. The VDR has been shown in muscle cells by immunohistochemistry, and the number of receptors decreases with aging [94]. vitamin D could still act on muscle through another receptor, through neuromuscular interaction, or through serum calcium. The active vitamin D metabolite 1,25(OH)2D stimulates differentiation of myoblasts [96]. Furthermore, it stimulates calcium influx, phosphate transport, and muscle fiber differentiation. 1,25(OH)2D may also bind to a membrane receptor, activating cyclic AMP or arachidonic acid. Subsequently, calcium is actively transported into the sarcoplasmic reticulum, increasing intracellular calcium, necessary for cross-bridge formation, and hence muscle contraction [97].

INTERVENTIONS IN SARCOPENIA:

Many study demonstrated an improvement in muscular function that includes strength and performance and also decrease the progression of sarcopenia with nutritional supplementation and exercise. Exercising programs need to be relevant, effective, safe and realistic for the frail elderly population they are targeted towards prevention of disability. Participation in

regular exercise programs requires motivation from the individual which may be difficult for some older subjects.

PHYSICAL EXERCISE:

	Recommendation
The American College of Sports Medicine The American Heart Association (Nelson et al., 2007)	Resistance exercise 8 – 10 exercises (using the major muscle groups) 10 – 15 repetitions Performed at 70-90% of one maximum repetition On 2 non-consecutive days per week
Society for Sarcopenia, Cachexia and Wasting Disease (Morley et al., 2010)	Combination of resistance and aerobic exercise 20-30 minutes exercise 3 times per week
Visvanathan and Chapman (2010)	Combination of resistance and aerobic exercise Minimum 50% resistance exercise 30-45 minutes exercise 3-5 times per week

NUTRITION SUPPLEMENTATION:

PROTEIN:

Many interventional study also suggest the nutritional supplementation in all sarcopenic elderly which shows a significant improvement in ahe muscle strength and performance. The effect will be maximal if it is along with the exercise program. Cambell et al study shows that elderly have high protein requirements to maintain muscle mass and function and the elderly have low protein intake so protein intake of 1.0-1.5g/kg/d is recommended in sarcopenic subjects.

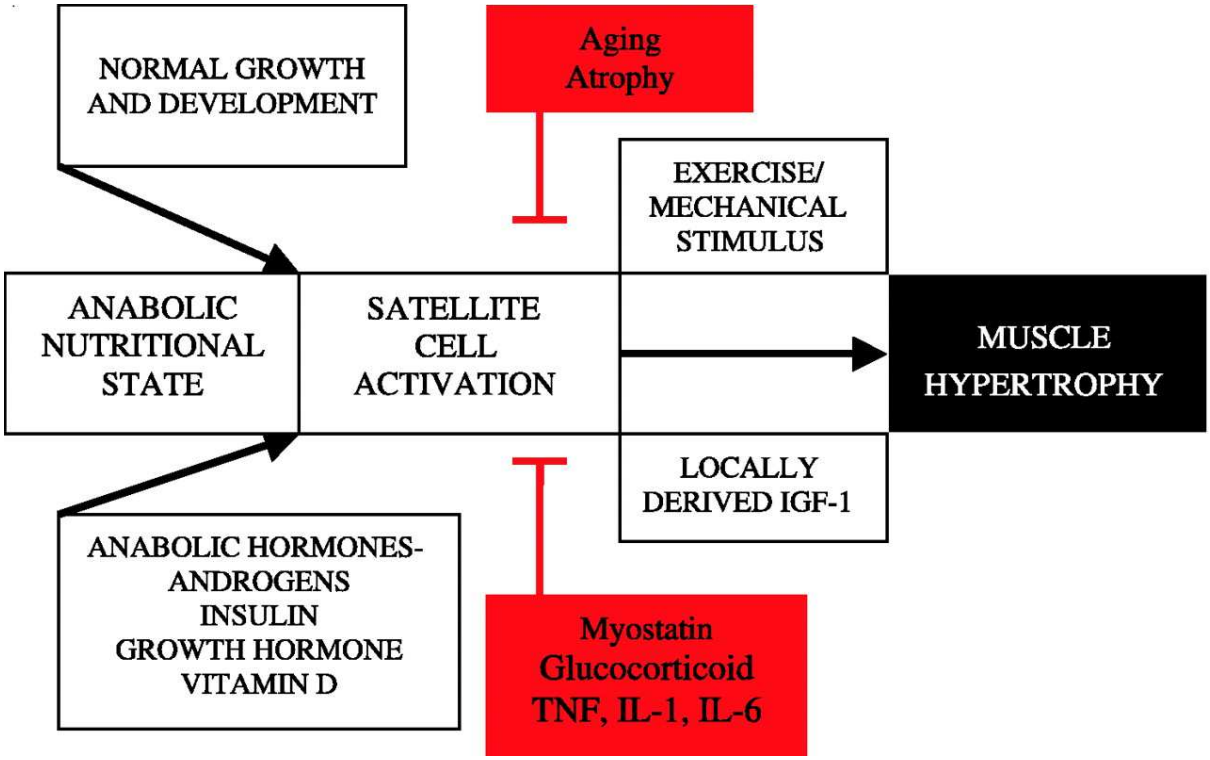
ESSENTIAL AMINOACID:

The availability of blood AAs is a potent stimulator of muscle protein synthesis. As the extraction of essential amino acid increases with ageing the availability of essential amino acid decreases. Studies have proven that 15g of essential amino acid increases muscle protein synthesis in both young and elderly population. Of EAAs, leucine is the most potent stimulator of muscle protein synthesis.

VITAMIN D:

Bischoff-Ferrari et al. studies shows 800IU vitamin D supplementation (2-12 months) improves lower extremity strength and function and balance.

The Society for Sarcopenia, Cachexia and Wasting Disease published nutritional recommendations for sarcopenia in 2010. This recommends a balanced protein intake and energy supplement may be useful in preventing and reversing sarcopenia as part of a multimodal therapeutic approach



SOCIETY OF SARCOPENIA CACHEXIA AND WASTING DISORDER RECOMMENDATION FOR SARCOPENIA:

- 15% to 38% of elderly male and 27% to 41% of elderly female intake is lower than the RDA for protein so the supplementation of protein and increase in dietary supplementation was recommended.

- It is recommends a protein intake of about 1 to 1.5 g/kg/day.

- It is recommended that a leucine rich diet and supplement should be added to the diet.

- Creatine may increase the effects of physical exercises in sarcopenic individual.

- 25 hydroxyD3 levels should be evaluatedin all elderly with sarcopenia.

- Vitamin D is recommended in doses needed to increase the levels above 100 nmol/L as an adjunctive therapy.

MATERIALS AND METHODS

MATERIALS AND METHODS

STUDY CENTRE:

Rajiv Gandhi Government General Hospital and Madras Medical College, Chennai.

ETHICAL COMMITTEE APPROVAL:

Ethical committee clearance obtained from Institutional Ethical Committee of MADRAS MEDICAL COLLEGE held on 03.06.2014.

STUDY DESIGN:

Hospital based observational study.

PERIOD OF STUDY:

3 months duration June 2014 to August 2014

STUDY POPULATION:

100 patients.

INCLUSION CRITERIA:

1. Age above 65 years.

2. Patient who are giving consent for the participation in the study.

EXCLUSION CRITERIA:

1. Patients with severe osteoarthritis / lower limb fracture / hemiplegia / upper limb and lower limb disability
2. Patient with severe cognitive impairment who cannot understand the test instruction or give informed consent.
3. Patients with diabetes mellitus / chronic kidney disease / COPD / Acute Illness.
4. Patient with severe malnourishment / cachexic / malignancy.
5. Patient taking vitamin D supplementation.

DETAILS OF THE STUDY:

100 Elderly population attending Geriatric medicine outpatient department in Rajiv Gandhi Government General Hospital were selected in the study as per above mentioned inclusion and exclusion criteria. Relevant history about co morbidities was obtained. Anthropometric evaluation was done. Muscle mass was evaluated using body composition analyzer. Muscle strength was assessed based on hand grip strength using handheld Dynamometer. The patient was asked to do for 6 times (3 times in each hand) and the greater value was taken as handgrip strength. Muscle performance was assessed based on gait speed using 4meter walk test. Out of 100 study

subjects, 50 healthy elderly without sarcopeniaselected as control group and 50 subjects with sarcopenia(based on EWGSOP definition) as cases. The Vitamin D level of both the group was assessed by means of serum 25, hydroxyl cholecalciferol, a surrogate marker of Vitamin D using chemiluminiscence method.

The vitamin D level of both the group with and without sarcopenia was assessed and analysed.

OBSERVATION AND RESULTS

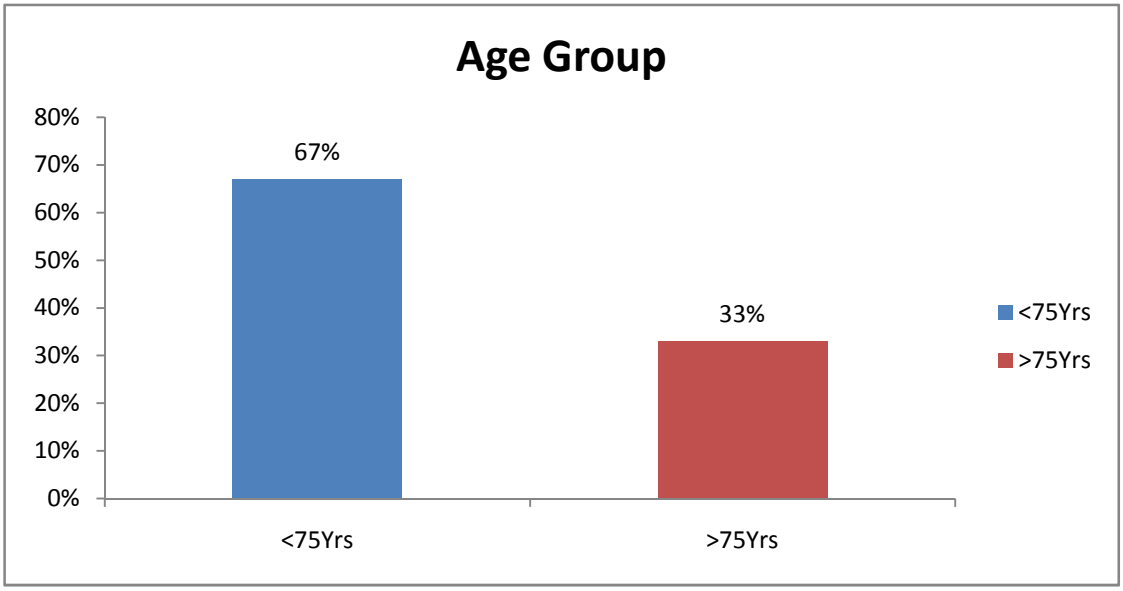
OBSERVATION AND RESULTS

A total of 100 elderly population of age greater than 65years was selected in our study. 50 elderly cases with sarcopenia based on the muscle mass, muscle strength and muscle performance and 50 elderly controls without sarcopenia was selected based on the inclusion and exclusion criteria as mentioned. In the 100 study population ,50 were male and 50 were female. The vitamin D level of both group was analysed and the results are as follows.

AGE DISTRIBUTION:

Age	Frequency	Percent
<75 Yrs	67	67.0
>75 Yrs	33	33.0
Total	100	100.0

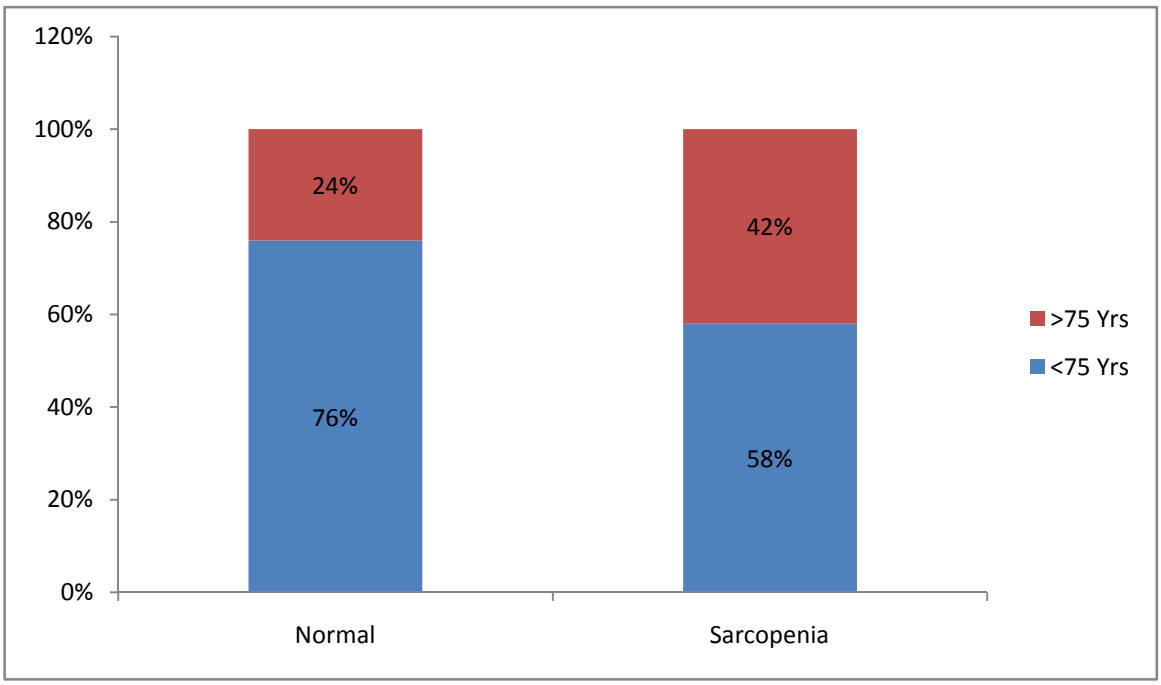
In our study 67% of population were between 65 – 75 yrs of age and 33% were more than 75 years of age.The mean age of the subjects in the study was 72.99 ± 4.56.



AGE DISTRIBUTION IN BOTH GROUP:

			GROUP		Total	CHI SQUARE	P VALUE
			NORMAL	SARCOPENIA			
AGEGR	<75 Yrs	Count	38	29	67	3.66	0.06
		% within AGEGR	76%	58.0%	100.0%		
	>75 Yrs	Count	12	24	33		
		% within AGEGR	24%	42.0%	100.0%		
Total		Count	50	50	100		
		% within AGEGR	50.0%	50.0%	100.0%		

In our study 76% were in age group ‧ 75 yrs and 24% were ‧ 75 yrs in the healthy control group. 58% were ‧ 75 yrs and 42% were ‧ 75 yrs in sarcopenic groups.

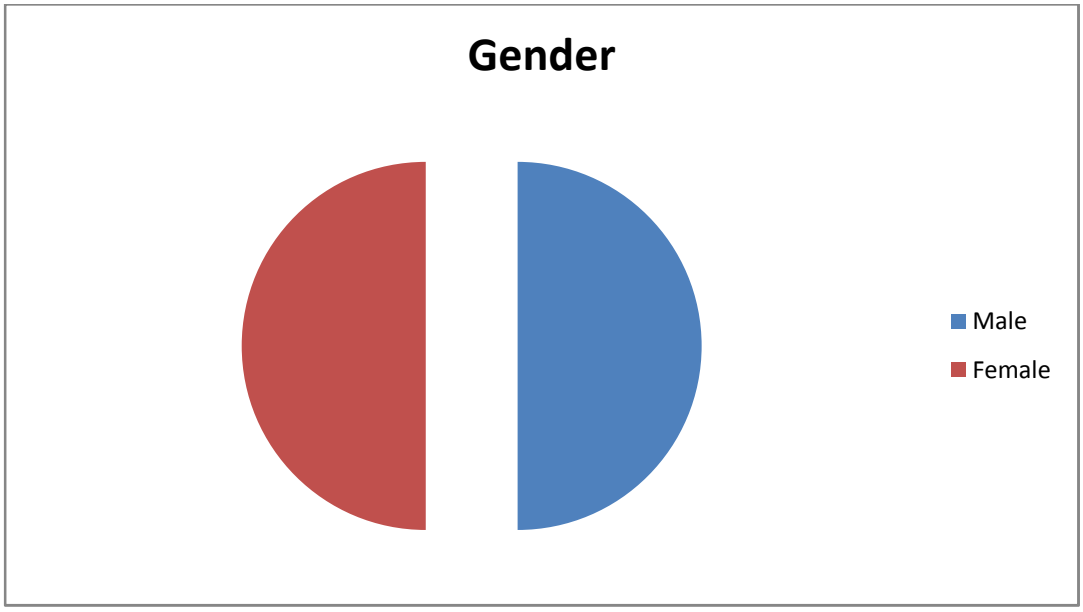


The age distribution between the two study groups doesn't show significant difference with p value 0.06(p▪ 0.05).

SEX DISTRIBUTION:

	Frequency	Percent
Male	50	50.0
Female	50	50.0
Total	100	100.0

Our study included of 100 subjects, out of which 50 were male and 50 were female. In control group and in sarcopenic group, 50% were male and 50% were female.



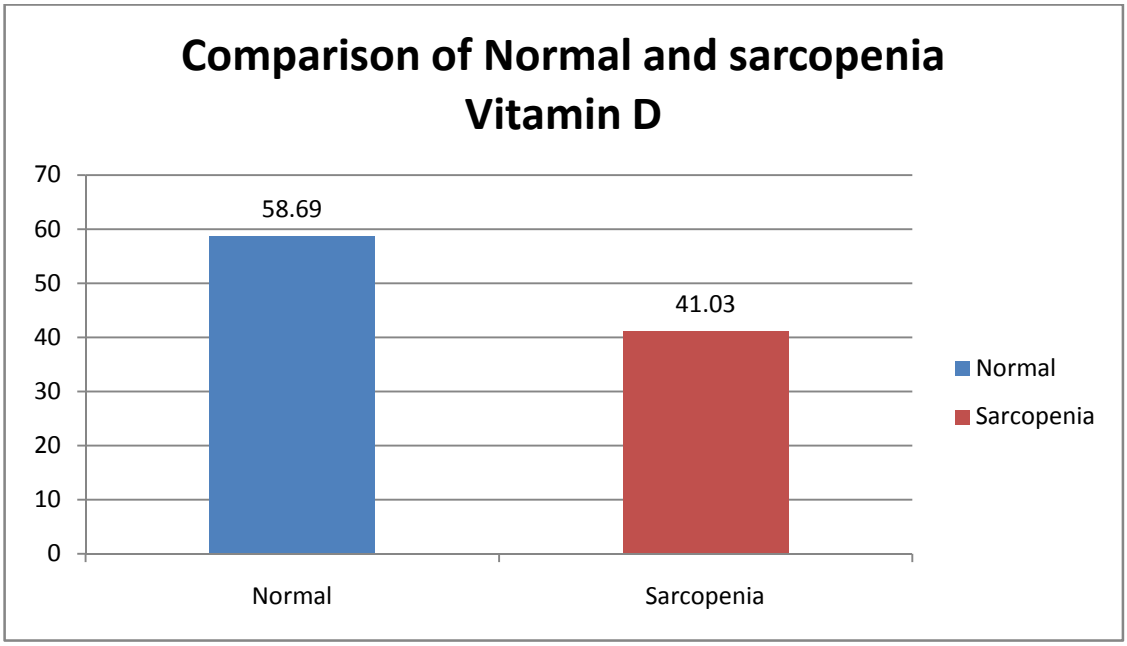
COMPARISON OF VITAMIN D LEVEL:

In our study the prevalence of vitamin D level in sarcopenic group was 86% and in healthy group was 78%.

	GROUP	Mean	Std. Deviation	Minimum	Maximum	Range
Vit D	NORMAL	58.69	16.36	22.13	86.44	64.31
	SARCOPENIA	41.03	16.75	23.45	80.13	56.68

The mean vitamin D in healthy group was 58.69±16.36nmol and in sarcopenic group was 41.03±16.75nmol.

T-TEST FOR COMPARISON OF NORMAL AND SARCOPENIA



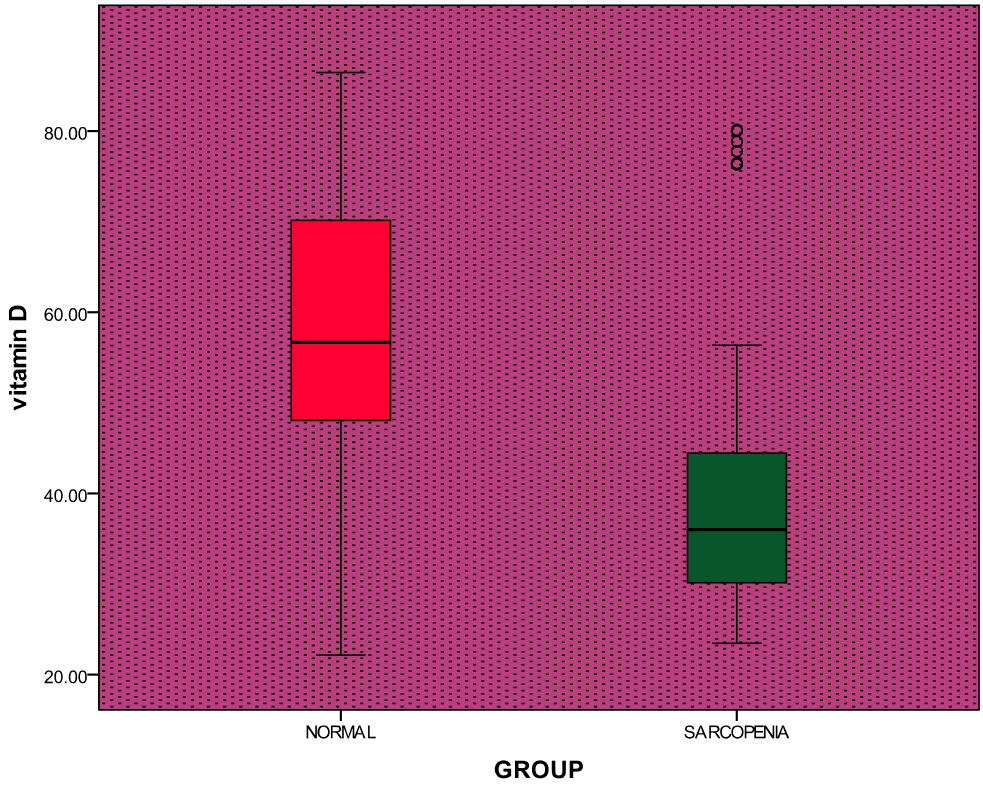
The mean level of Vitamin D [25(OH) D3] shows a significant difference with mean difference of 17.66 nmol.

	t-test for Equality of Means						
	t	df	PValue	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
						Lower	Upper
VIT D	5.334*	98	.001	17.66220	3.31107	11.09150	24.23290

*significant at P<0.05

The Highest vitamin D level86.44nmol and the lowest value of 22.13nmol in healthy group.The highest value 80.13nmol and 23.45nmol lowest value 23.45nmol in sarcopenic group.

VITAMIN D AND SARCOPENIA



The mean value in Vitamin D level of the two groups shows a significant difference with p = 0.001 using Chi Square test.

COMPARISON OF SARCOPENIA AND SEVERE SARCOPENIA:

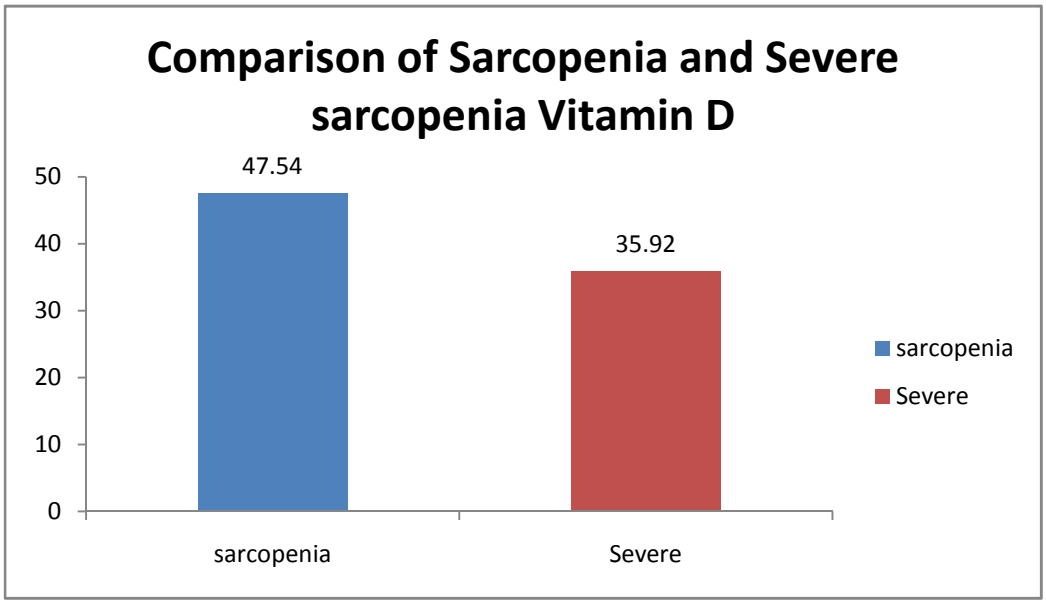
The vitamin D level of sarcopenic and severe sarcopenic were compared.

	sarcopenia group	N	Mean	Std. Deviation	Minimum	Maximum	Range
vitD	Sarcopenia	22	47.54	20.21	24.98	80.13	55.15
	Severe sarcopenia	28	35.92	11.41	23.45	76.45	53.00

The mean vitamin D level in sarcopenic and severe sarcopenic were 47.54 ± 20.21nmol and 35.92 ± 11.41 nmol respectively.

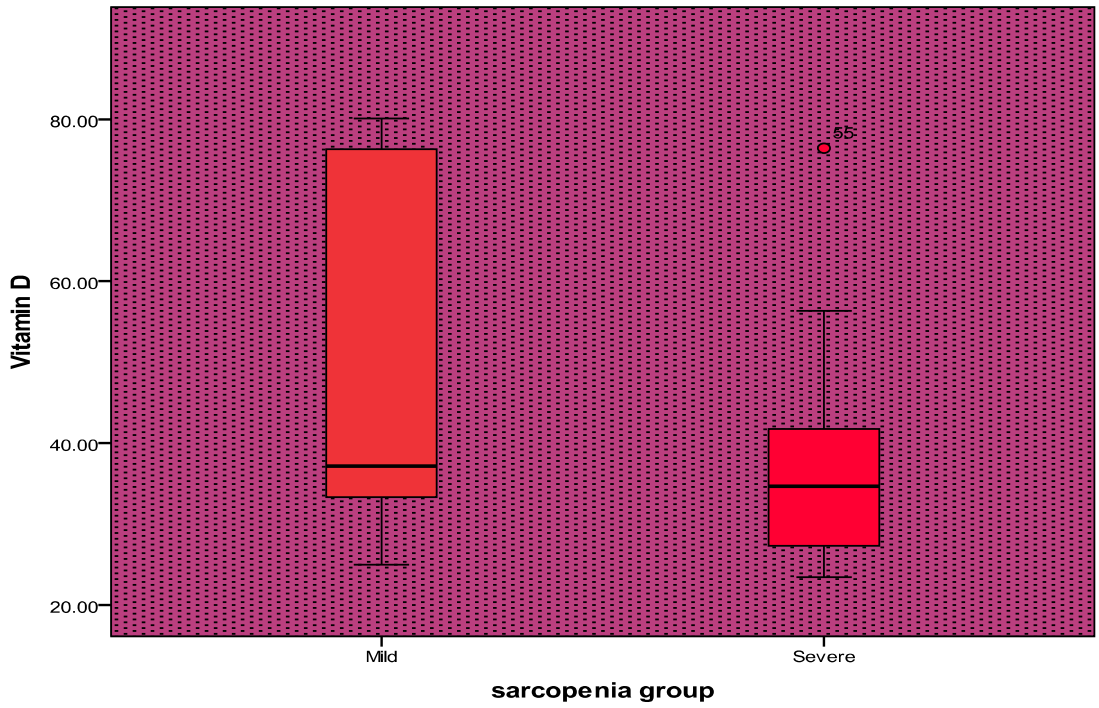
	t-test for Equality of Means						
	t	df	P Value	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
						Lower	Upper
Vitamin D	2.569*	48	.013	11.61519	4.52125	2.52462	20.70577

*significant at P<0.05



The mean difference in vitamin D level of sarcopenic and severe sarcopenic was 11.61nmol.UsingChi Square test the mean value show a significant difference with p value ▪ 0.05

VITAMIN D LEVEL AND SARCOPENIA GROUP

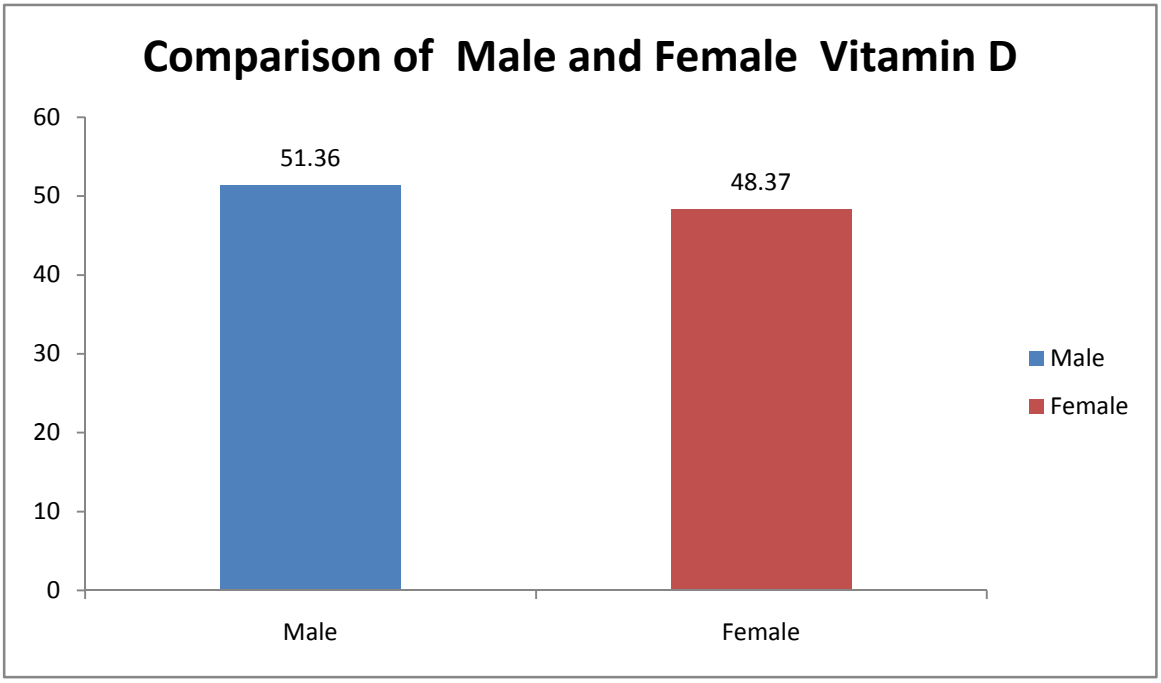


VITAMIN D LEVEL IN MALE AND FEMALE:

T-Test FOR VITAMIN D FOR SEX:

			Statistic				
		N	Mean	Std. Deviation	Minimum	Maximum	Range
Vit D	Male	50	51.36	18.66	23.50	86.44	62.94
	Female	50	48.37	18.83	22.13	85.46	63.33

In our study, the mean vitamin D level of male was51.36 ± 18.66nmol and female was 48.37 ± 18.83 nmol.

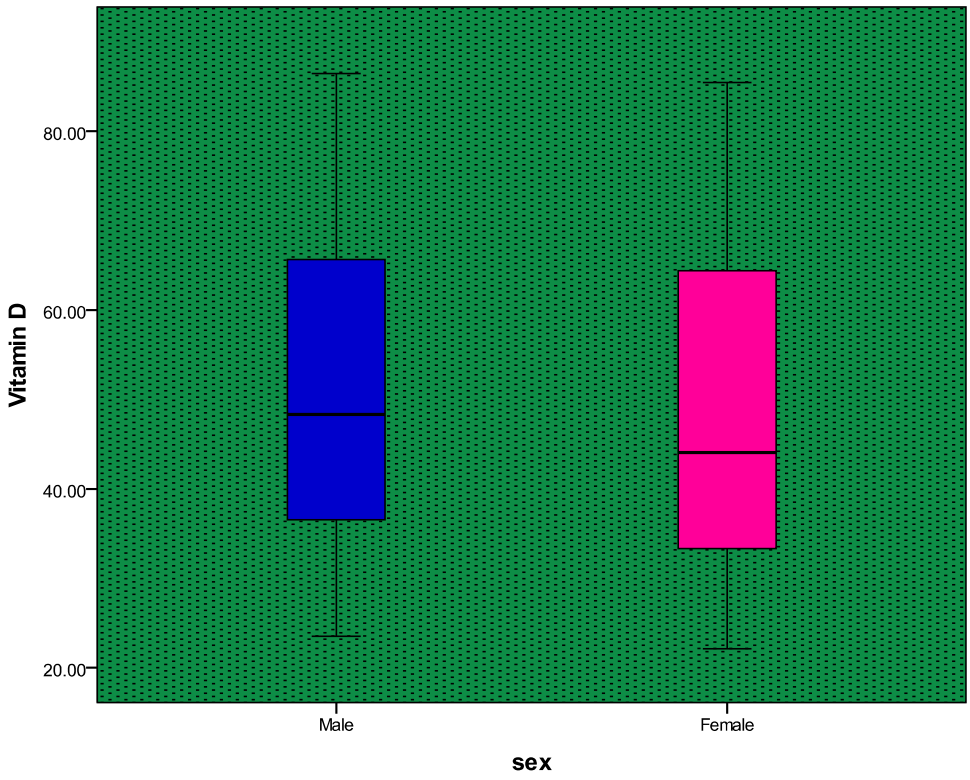


Comparison of male and female vitamin D:

	t-test for Equality of Means						
	t	df	P Value	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
						Lower	Upper
Vitamin D	0.798	98	.427	2.99300	3.74899	-4.44675	10.43275

The mean difference in Vitamin D level of male and female doesn't show a significant difference with p value ▪ 0.05.

VITAMIN D - MALE AND FEMALE

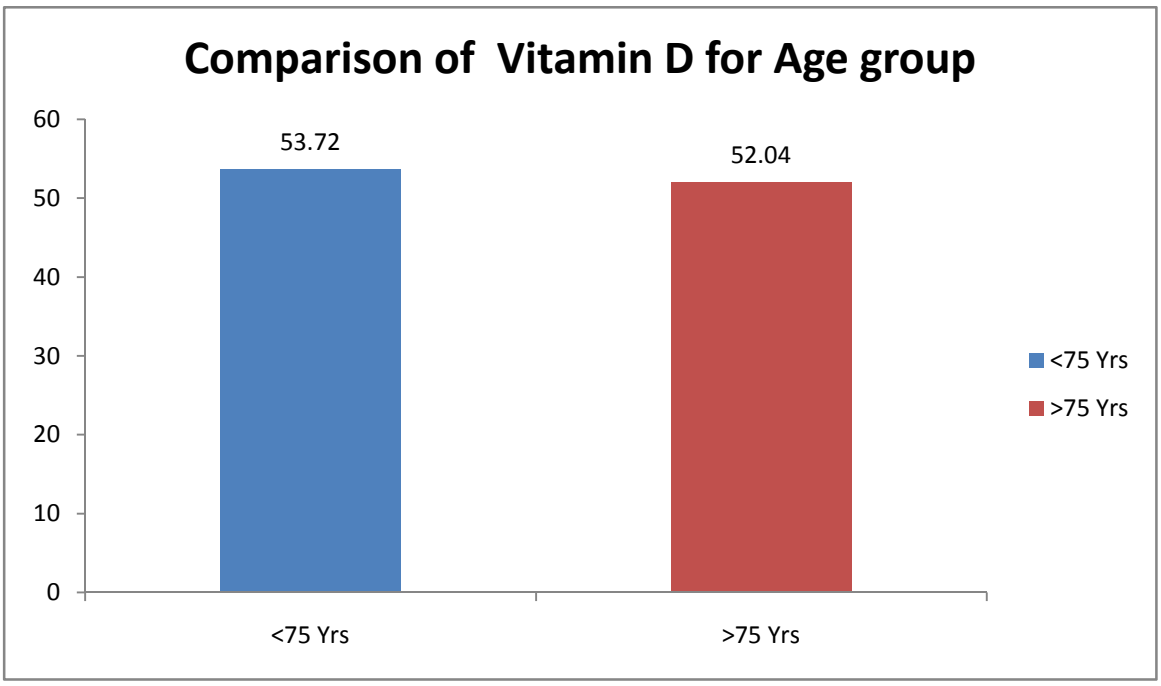


COMPARISON OF VITAMIN D FOR AGE GROUP:

T-Test FOR VITAMIN D FOR Age group:

	AGEGR	N	Mean	Std. Deviation	Minimum	Maximum	Range
Vit D	<75 Yrs	67	53.72	19.00	22.13	86.44	64.31
	>75 Yrs	33	52.04	15.63	23.45	80.01	56.56

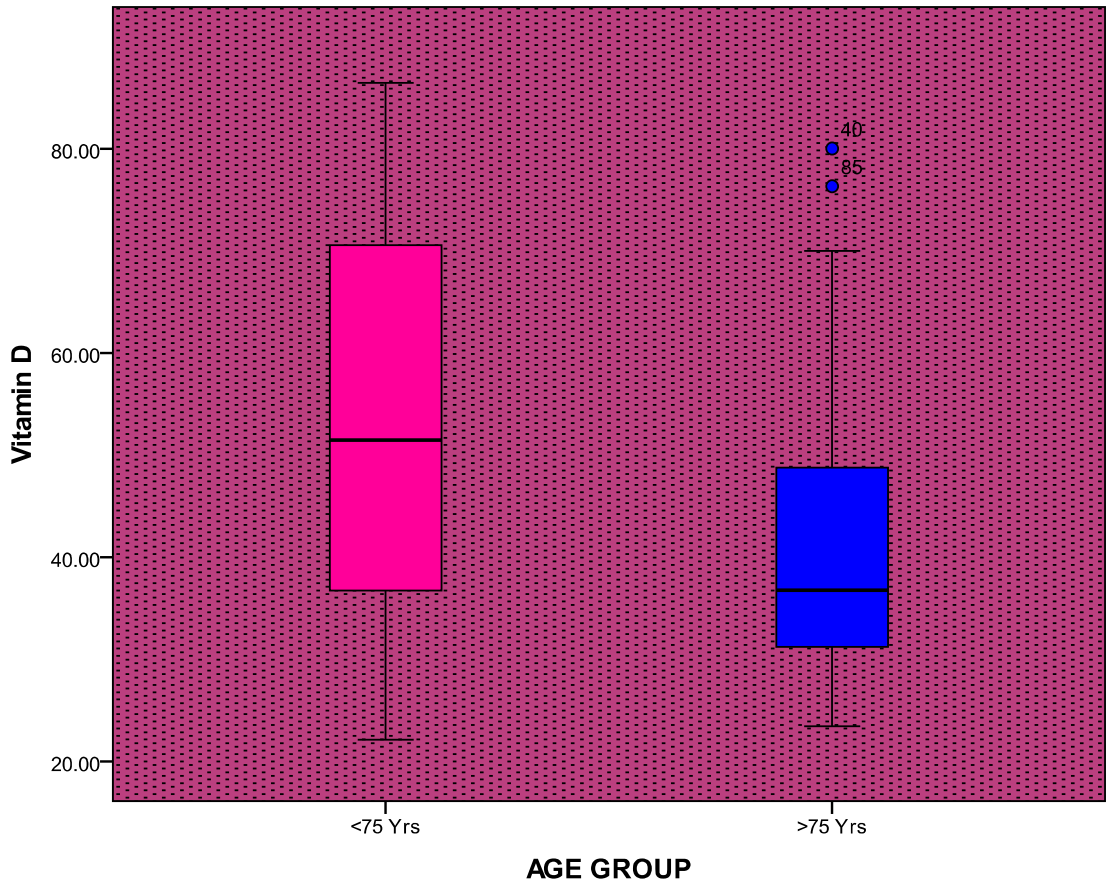
The mean Vitamin D value of both age group 65 – 75 yrs and more than 75 yrs were 53.72 ± 19nmol and 52.04 ± 15.63 nmol respectively.



	t-test for Equality of Means						
	t	df	P Value	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
						Lower	Upper
VitaminD	0.470	98	.32	1.68	3.82152	0.98	2.38

The mean difference in vitamin D level in the two age group show no significant difference with p value ▪ 0.05.

PREVALENCE OF VITAMIN D – AGE GROUP



PREVALENCE OF VITAMIN D:

The prevalence of vitamin D in both study group of healthy and sarcopenic elderly were 78% and 86% respectively.

VITAMIN D GROUP

	Frequency	Percent	Valid Percent	Cumulative Percent
Deficiency	9	9.0	9.0	9.0
Insufficiency	73	73.0	73.0	82.0
NORMAL	18	18.0	18.0	100.0
Total	100	100.0	100.0	

In our study out of 100 subjects[including both healthy and sarcopenic] 9 subjects had severe deficiency, 73 had insufficient levels and 18 had normal range Vitamin D level.

NORMAL VITAMIN D:

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
NORMAL	18.00	79.93	3.59	0.85	78.14	81.71	75.87	86.44

VITAMIN D INSUFFICIENCY:

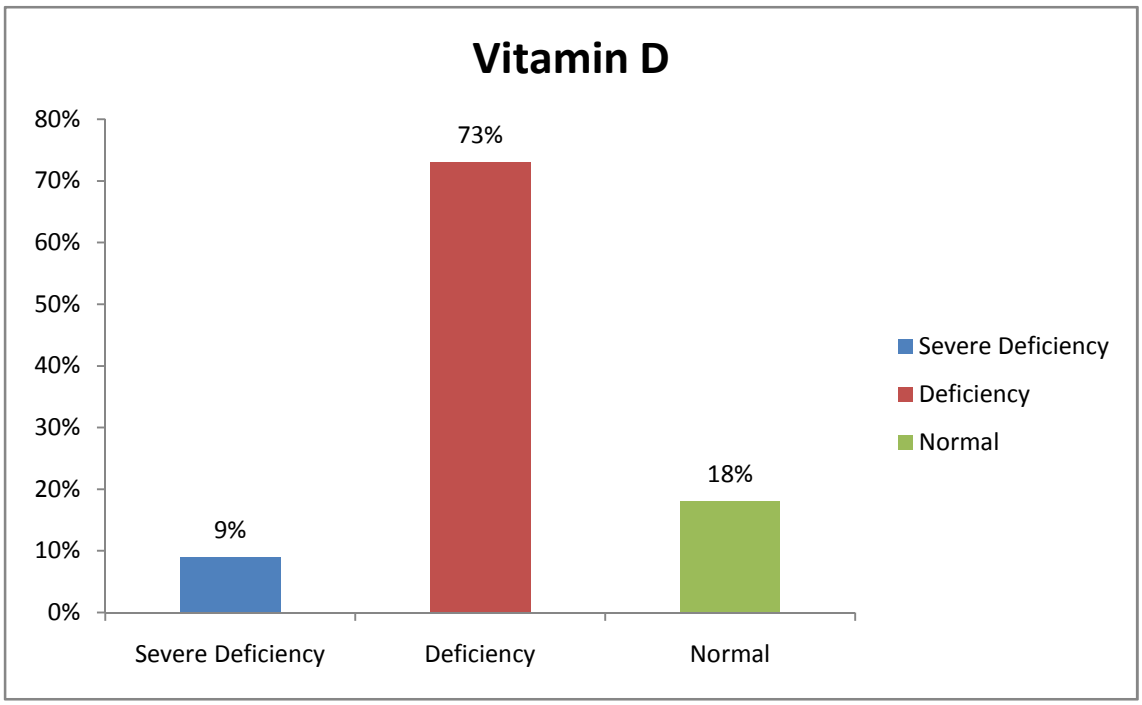
	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
INSUFFICIENCY	73.00	45.64	12.27	1.44	42.77	48.50	25.65	71.00

The mean Vitamin D level in subjects with insufficiency was 45.64 ± 12.27 nmol; the highest value was 71 nmol and lowest value was 25.65 nmol.

VITAMIN D DEFICIENCY:

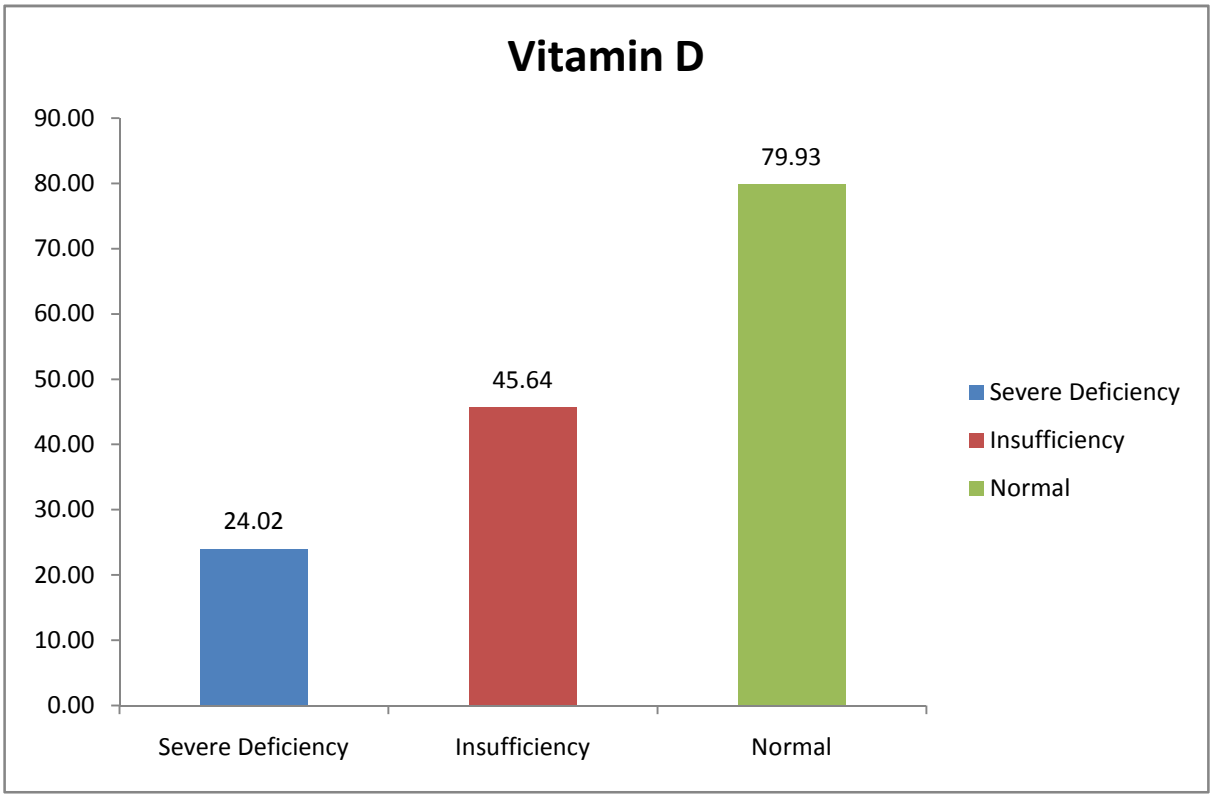
	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
SEVERE DEFICIENCY	9.00	24.02	0.94	0.31	23.30	24.74	22.13	25.00

PREVALENCE OF VITAMIN D GROUPS



The mean vitamin D level in deficiency, insufficiency and normal were 24.02nmol, 45.64nmol, and 79.93nmol respectively.

MEAN VITAMIN D LEVEL



VITAMIN D DEFICIENCY AND STAGES OF SARCOPENIA:

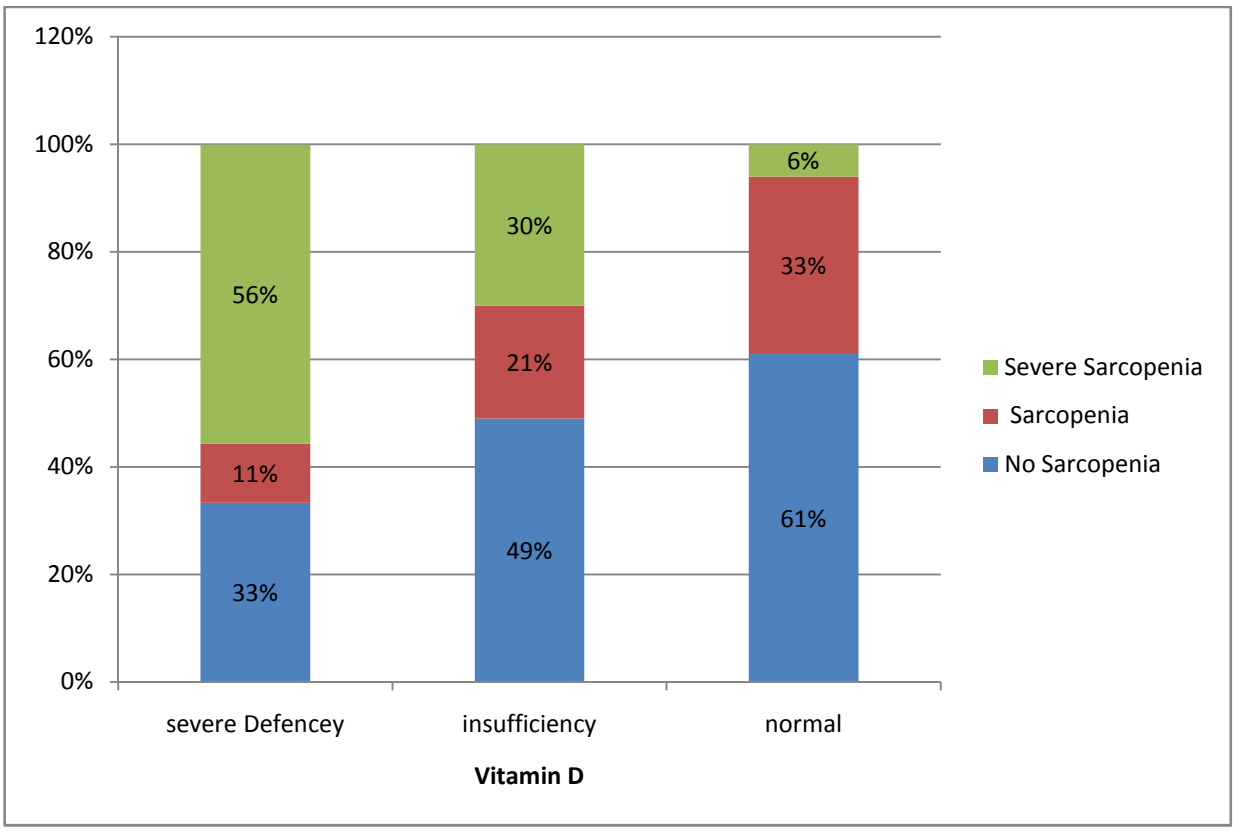
VITAMIN D AND STAGES OF SARCOPENIA

			Group			
			no	Sarcopenia	Severe sarcopenia	Total
VITAMIN D GROUP	DEFICIENCY	Count	3	1	5	9
		% within VITAMIN D GROUP	33.3%	11.1%	55.6%	100.0%
	INSUFFICIENCY	Count	36	15	22	73
		% within VITAMIN D GROUP	49.3%	20.5%	30.1%	100.0%
	NORMAL	Count	11	6	1	18
		% within VITAMIN D GROUP	61.1%	33.3%	5.6%	100.0%
Total		Count	50	22	28	100
		% within VITAMIN D GROUP	50.0%	22.0%	28.0%	100.0%

In our study 18% had normal vitamin D status in which 61.1% subjects were without sarcopenia, 33.3% were sarcopenic and 5.6% were severe sarcopenic. In the study group ,73% had insufficient Vitamin D status in which 49.3% subjects were without sarcopenia; 20.5% with sarcopenia and 30.1% with severe sarcopenia.

In our study 9% of the study subjects were severely Vitamin D deficient, out of which 33.3% had no sarcopenia, 11.1% had sarcopenia and 55.6% had severe sarcopenia.

VITAMIN D AND STAGES OF SARCOPENIA



The frequency of occurrence of severe vitamin D deficiency show a significant difference comparing with the sarcopenic group and without sarcopenic group.

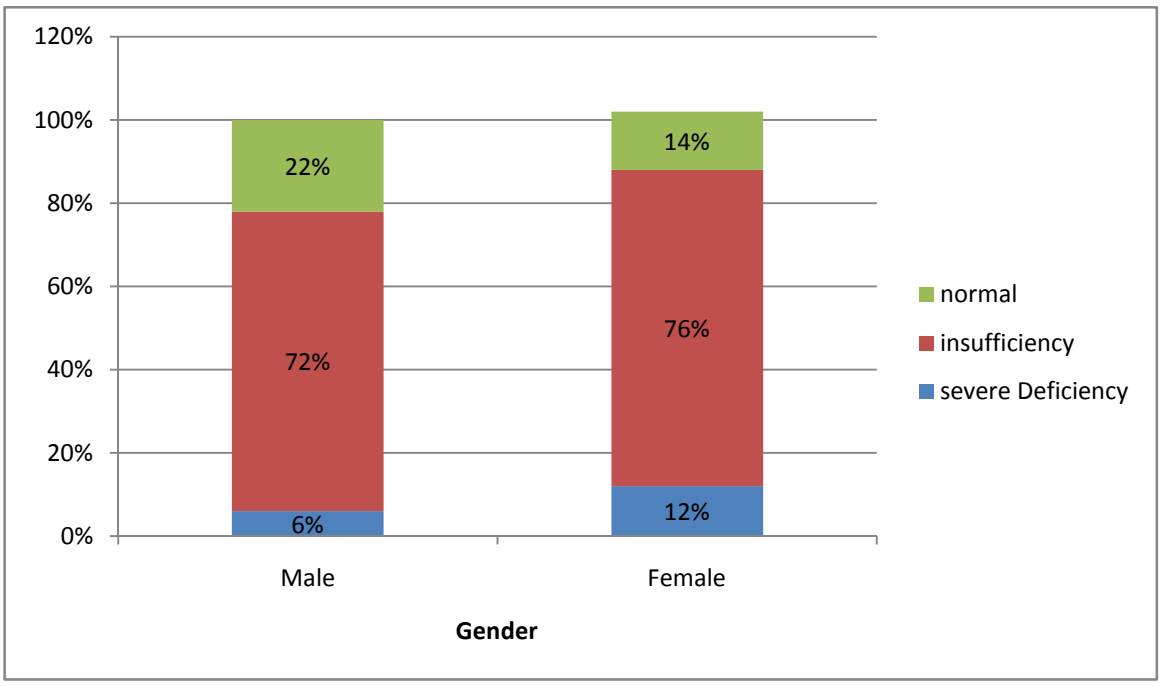
LEVELS OF VITAMIN D AND SEX DISTRIBUTION:

VITAMIN D GROUP AND SEX DISTRIBUTION

			Sex		Total
			Male	Female	
VITAMIN D GROUP	DEFICIENCY	Count	3	6	9
		% within VITAMIN D GROUP	33.3%	66.7%	100.0%
	INSUFFICIENT	Count	36	37	73
		% within VITAMIN D GROUP	49.3%	50.7%	100.0%
	NORMAL	Count	11	7	18
		% within VITAMIN D GROUP	61.1%	38.9%	100.0%
Total		Count	50	50	100
		% within VITAMIN D GROUP	50.0%	50.0%	100.0%

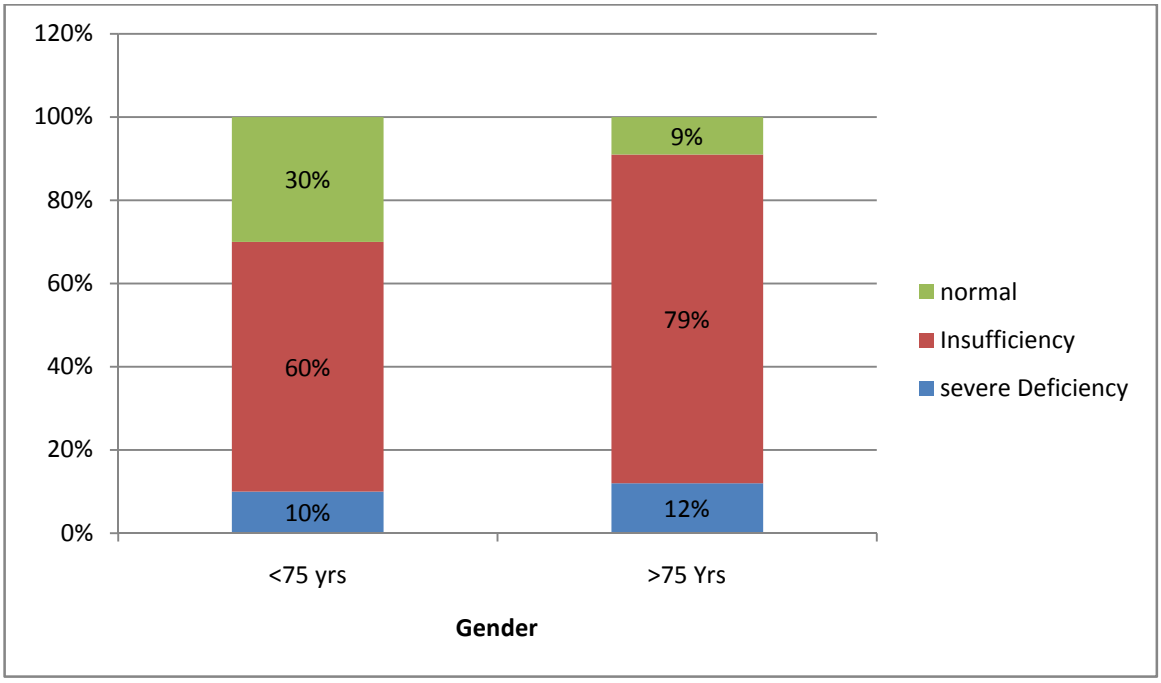
In our study, out of 50 male subjects 11 had normal vitamin D status, 36 had insufficient levels and 33.3 were deficient. Out of 50 female subjects in our study 7 had normal Vitamin D level status37 had insufficient levels and 6 were deficient.

LEVELS OF VITAMIN D AND SEX DISTRIBUTION



The vitamin D level in male and female group show no significant difference using chi square test statistical analysis.

VITAMIN D GROUP AND AGE DISTRIBUTION



VITAMIN D GROUP AND AGE DISTRIBUTION CROSS TABLE:

Crosstab							
			AGE		Total	Chi square	P Value
			<75 Yrs	>75 Yrs			
VITAMIN D GROUP	deficiency	Count	5	4	9	0.231	0.89
		% within VITAMIN D GROUP	55.56%	44.44%	100.00%		
	insufficiency	Count	47	26	73		
		% within VITAMIN D GROUP	64.38%	35.62%	100.00%		
	NORMAL	Count	15	3	18		
		% within VITAMIN D GROUP	83.33%	16.67%	100.00%		
Total		Count	67	33	100		
		% within VITAMIN D GROUP	67.00%	33.00%	100.00%		

In our study the vitamin D level in the age group <75 years and > 75 years, show no significant difference with p value ▪ 0.05 using Chi Square test of statistical analysis.

VITAMIN D AND HAND GRIP STRENGTH:

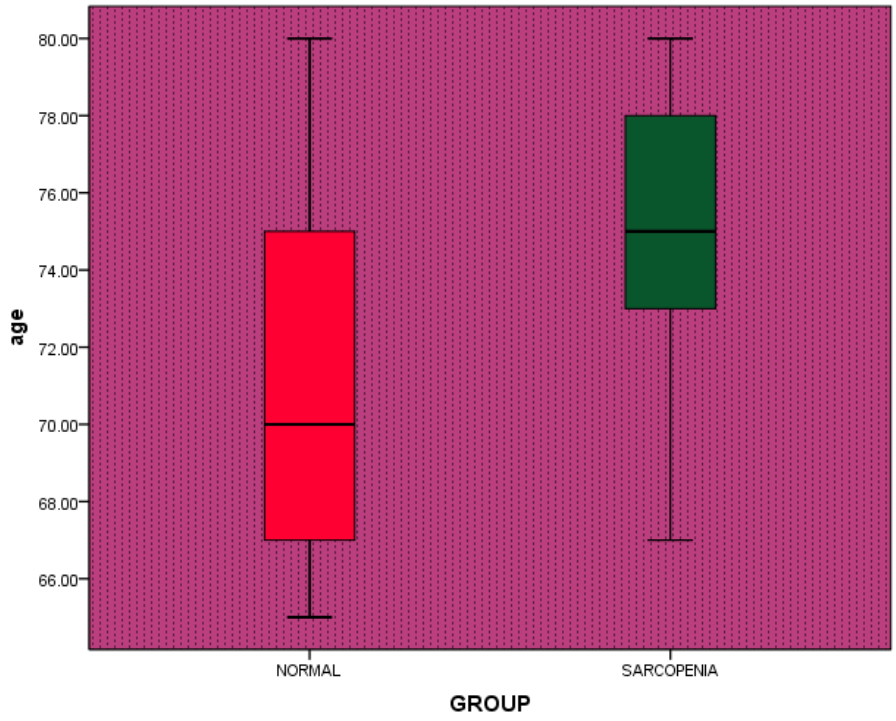
Descriptive Statistics

	N	Minimum	Maximum	Mean	Std. Deviation
age	100	65.00	80.00	72.99	4.55
HAND_GRIP	100	1.00	50.00	22.53	13.18
GAIT_SPEED_MSEC	100	0.30	1.20	0.80	0.18
N	100				

The mean hand grip strength in normal study group was 32.62±8.70kg and the mean hand grip strength in sarcopenic was 12.44±8.21kg. The mean gait speed in normal and sarcopenic individual was 0.89 and 0.69 m/sec.

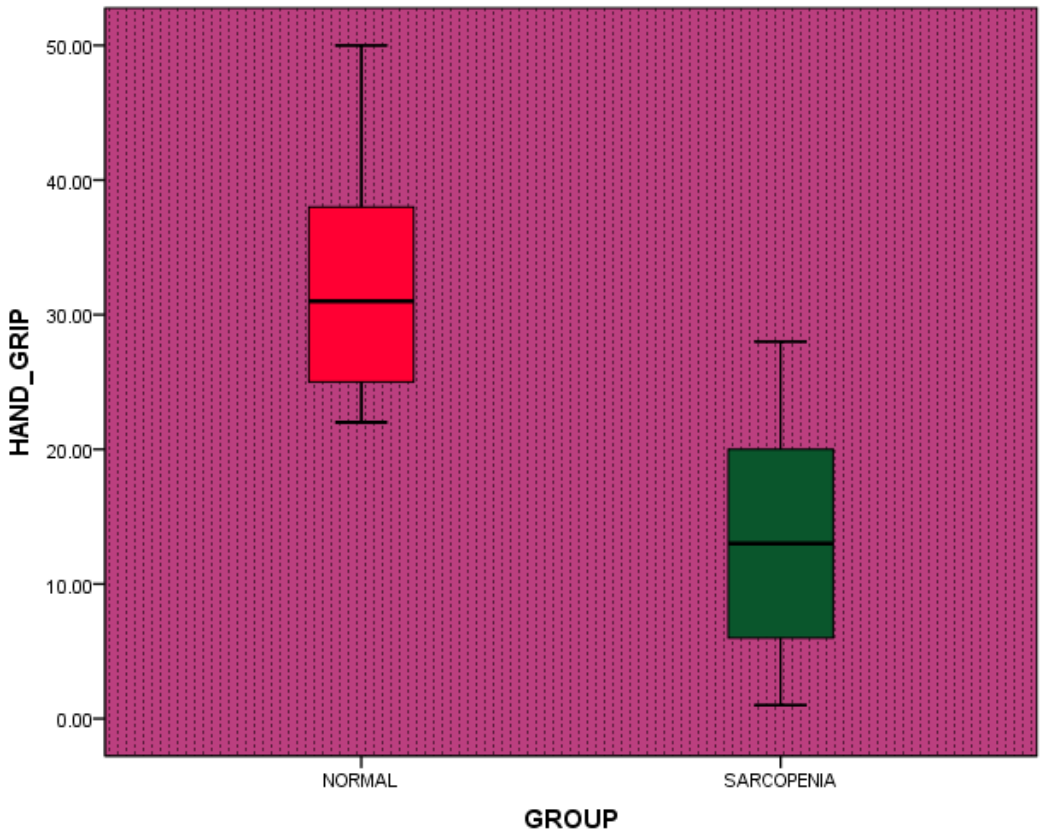
	group	Mean	Std. Deviation	Minimum	Maximum	Range
age	NORMAL	71.00	4.58	65.00	80.00	15.00
	SARCOPENIA	74.98	3.55	67.00	80.00	13.00
HAND_GRIP	NORMAL	32.62	8.70	22.00	50.00	28.00
	SARCOPENIA	12.44	8.21	1.00	28.00	27.00
GAIT_SPEED_MSEC	NORMAL	.89	.08	.81	1.12	.31
	SARCOPENIA	.69	.19	.30	1.20	.90

SARCOPENIA AND AGE



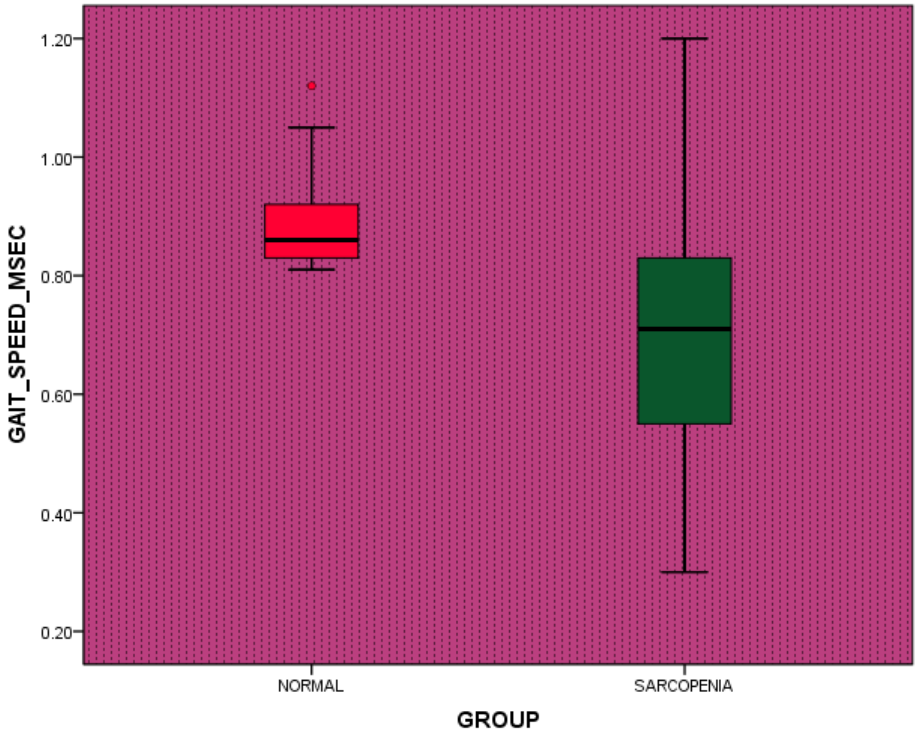
Mean age of the subjects without sarcopenia was 71±4.58 and with sarcopenia was 74.98±3.55. Mean hand grip strength in subjects without sarcopenia was32.62±8.70kg and with sarcopeniawas 12.44±8.21kg.

SARCOPENIA AND HAND GRIP



Average gait speed in subjects without sarcopenia was 0.89±0.08m/sec and in subjects with sarcopenia was 0.69±0.19m/sec.

SARCOPENIA AND GAIT SPEED

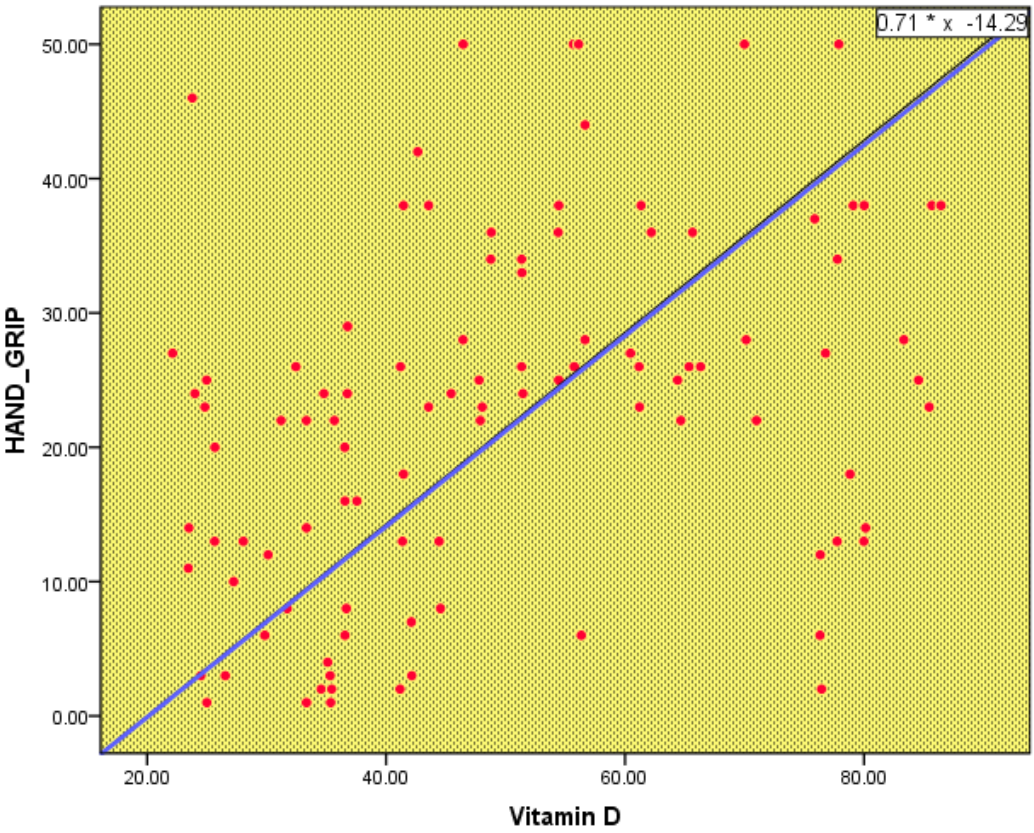


ASSOCIATION BETWEEN VITAMIN D AND HANDGRIP STRENGTH:

Correlations			
	HAND_GRIP		
	Pearson Correlation	P Value	N
HAND_GRIP Vs Vitamin D	.372**	.0001	100

Hand grip strength and vitamin D level show a significant association using pearson correlation with p ▪ 0.0001.

ASSOCIATION BETWEEN VITAMIN D AND HANDGRIP STRENGTH



DISCUSSION

DISCUSSION

Sarcopenia is a complex multifactorial condition that can be treated with multimodal approaches. Although there is a large body of epidemiologic evidence linking sarcopenia to loss in physical function and morbidity, the assessment and management of saropenia are not a routine part of clinical practice. This study is a small effort to popularise the consensus definition of sarcopenia and its relation with vitamin D level, one of the contributing factor of sarcopenia.

Our study consists of 100 participants with 50 as a control group without srcopenia and 50 as study group with sarcopenia based on EWGSOP definition. The prevalence of vitamin D deficiency/insufficiency among the control group was 78% and among the study group was 86%. According to a study regarding prevalence of osteoporosis in Southern India conducted by Harinaryanan et al, the prevalence of Vitamin D deficiency / insufficiency was about 50-90%. In North India, studies conducted by Goswami et al, Arya et al, the prevalence was about 78-96%. These studies were conducted in healthy elderly and healthy adults .

In our study population the prevalence of Vitamin D deficiency/insufficiency in male and female was 78% and 86% respectively. The male and female were almost equally affected. These results correspond to the study result of Mizumoto et al [11]

Many studies demonstrated Vitamin D level and its association with skeletal muscle mass, strength and performance in the form of walking speed, balance and swaying [98 - 100].The Third National Health and Nutrition Examination Survey (NHANES III) study with 4100 participant showed a reduced muscle function in subjects with low vitamin D level. This decline in muscle function may be due to atrophied type II fibre in vitamin D deficiency [101].performance tends to improve within the reference range of 22.5 to 95 nmol/L, with the most dramatic improvements occurring in the range of 22.5 to 40 nmol/L [98].

The Longitudinal Study of Aging Amsterdam (LASA) study also demonstrates a negative correlation between muscle performance (Mass and Hand grip strength) and Vitamin D level. A more recent longitudinal study, the Cardiovascular Health Study also shows similar results [104 - 106]

In our study though the prevalence of Vitamin D in both the groups doesn’t show significant difference as the prevalence rate in healthy population was high, the mean 25,hydroxyl vitamin D3 level (the surrogate marker of VitaminD) in both the control and study group shows significant difference. The mean vitamin D3 level of control group without sarcopenia was 41.03± 16.75 nmol and that of subject group with sarcopenia was 58.69± 16.36 nmol and the mean difference in vitamin D level were 17.66nmol with significant p value of ▪ 0.05 comparing with the The Third National Health

and Nutrition Examination Survey NHANESIII and Longitudinal Study of Aging Amsterdam LASA study.

In our study the subject with severe sarcopenia i.e. with decreased muscle mass, muscle strength and physical performance has a mean Vitamin D level of 35.92±11.41nmol compared with sarcopeniai.e, with decreased muscle mass with decreased muscle strength or physical performance 47.54±20.21nmol. This level correlates with NHANES Study.

The prevalence of severe vitamin D deficiency in study population with sarcopenia was 33.3% compared to prevalence in population without sarcopenia of 11.1%..The mean vitamin D level of study population with severe sarcopenia was 35.92±11.41nmol compared to 58.69±16.36 in healthy elderly. .

In our study 9% of the study subjects were severely Vitamin D deficient, out of which 33.3% with no sarcopenia, 11.1% with sarcopenia and 55.6% were severe sarcopenic. The frequency of occurrence of severe vitamin D deficiency show a significant difference compare to the sarcopenic group and without sarcopeniagroup.This statistical results shows that sarcopenia even though a multifactorial syndrome has a negative correlation with the 25 Hydroxycholecalciferol level.

A study by Cambell et al recommends that the elderly have high protein requirements to maintain muscle mass and function. The geriatric population will have low intake of protein. A protein allowance of 1.0-1.5g/kg/d is recommended in sarcopenic subjects [109]. 15g Essential amino acid stimulates muscle protein synthesis in the young and elderly. Of EAAs, Leucine is the most potent stimulator of muscle protein synthesis. The Society for Sarcopenia, Cachexia and Wasting Disease published nutritional recommends the supplementation of Vitamin D to maintain Vitamin D level at 100nmol/L. Our study also shows a positive correlation of muscle performance (Handgrip strength) with vitamin D level using pearson's correlation formula.

Thus our study demonstrates an association between Vitamin D level and Sarcopenia and also with physical performance (Hand Grip strength). There is no significant difference between Male/Female and age group more than 75 and less than 75 years of age.

LIMITATIONS

LIMITATIONS

1. Duration of the study is only 3 months.

2. The study population includes only 100 subjects.

3. The outcome will be better in interventional and follow up study.

CONCLUSION

CONCLUSION

Sarcopenia is a age related decline in muscle mass and performance

The prevalence rate of vitamin D deficiency 78% in normal elderly and 86% in sarcopenic elderly. The prevalence shows no significant difference in both the study groups.

The mean difference in Vitamin D level of sarcopenic and normal study groupis 17.66nmol which indicates a significant difference. The mean difference in Vitamin D level of sarcopenic and severe sarcopenic is 11.62nmol which also shows a significant difference.

The male and female study population show no significant difference in their Vitamin D level. There is no significant difference in the Vitamin D levels between the age groups less than and more than 75 years.

The sarcopenic patients with vitamin D deficiency show a significantly low hand grip strength.

Thus sarcopenia, the cornerstone for frailty, and related to many adverse outcomes in the geriatric population is a multifactorial syndrome(one of the factors is Vitamin D which shows a negative correlation in our study). Sarcopenia can be managed with nutritional supplement and exercise programme which can improve the quality of life and functional independence.

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ANNEXURES

ABBREVIATIONS

ADL	-	Activity of daily living.
COPD	-	Chronic obstructive pulmonary disease.
EWGSOP	-	The European Working Group on Sarcopeniain older people.
DEXA	-	Dual energy X ray absorptiometry
BIA	-	Bio impedence analysis
TBK	-	Total body potassium
PBK	-	Partial body potassium
PEF	-	Peak expiratory flow
SPPB	-	Short physical performance battery
UV RAYS	-	Ultra violet rays
PCT	-	Proximal convoluted tubule
DCT	-	Distal convoluted tubule
PTH	-	Para thyroid hormone
VDR	-	Vitamin D receptor
CLIA	-	Chemiluminiscentimmuno assay
RDA	-	Recommended daily allowance
EAA	-	Essential amino acid

PROFORMA

A STUDY ON CORRELATION BETWEEN SARCOPENIA AND VITAMIN-D LEVEL IN ELDERLY

NAME :

AGE : SEX: OP NO. :

ADDRESS :

RELEVANT HISTORY:

1. Diabetic Mellitus:
2. History of fractures:
3. History of Malignancy:
4. History of Arthritis:
5. History of Stroke:
6. COPD:
7. Chronic Kidney Disease:
8. History of Vitamin D supplementation:

MUSCLE MASS %: HEIGHT : WEIGHT : BMI :

SKELETAL MUSCLE INDEX :

4METER GAIT SPEED (inmetre/ seconds) :

HAND GRIP STRENGTH (By hand dynamometer in kg):

TICKWHICHEVER IS APPLICABLE:

- PRESARCOPENIA ☐
- SARCOPENIA ☐
- SEVERE SARCOPENIA ☐
- NO SARCOPENIA ☐

VITAMIN D LEVEL:

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI-3

EC Reg No.ECR/270/Inst./TN/2013
Telephone No : 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr. S. Prabhagaran,
Post Graduate,
Department of Geriatric Medicine,
Madras Medical College, Chennai – 600003.

Dear Dr. S. Prabhagaran,

The Institutional Ethics Committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled **"Correlation between Sarcopenia and Vitamin-D level in elderly population"** No.39062014

The following members of Ethics Committee were present in the meeting held on 03.06.2014 conducted at Madras Medical College, Chennai-3.

1.	Dr. C. Rajendran, M.D.	-- Chairperson
2.	Dr. R. Vimala, M.D., Dean, MMC, Ch-3.	-- Deputy Chair Person
3.	Prof. Kalaiselvi, MD., Vice-Principal, MMC, Ch-3	-- Member
4.	Prof. Nandhini, M.D. Inst. of Pharmacology, MMC, Ch-3.	-- Member
5.	Dr. G. Muralidharan, Director Incharge , Inst. of Surgery	-- Member
6.	Prof. Md Ali, MD., DM., Prof & HOD of MGE, MMC, Ch-3.	-- Member
7.	Prof. Ramadevi, Director i/c, Biochemistry, MMC,Ch-3.	-- Member
8.	Prof. Saraswathy, MD., Director, Pathology, MMC, Ch-3.	-- Member
9.	Prof. Tito, Director, i/c. Inst. of Internal Medicine, MMC	-- Member
10.	Thiru. Rameshkumar, Administrative Officer	-- Lay Person
11.	Thiru. S. Govindasamy, BABL, High Court, Chennai-1.	-- Lawyer
12.	Tmt. Arnold Saulina, MA MSW	-- Social Scientist

We approve the proposal to be conducted in its presented form.

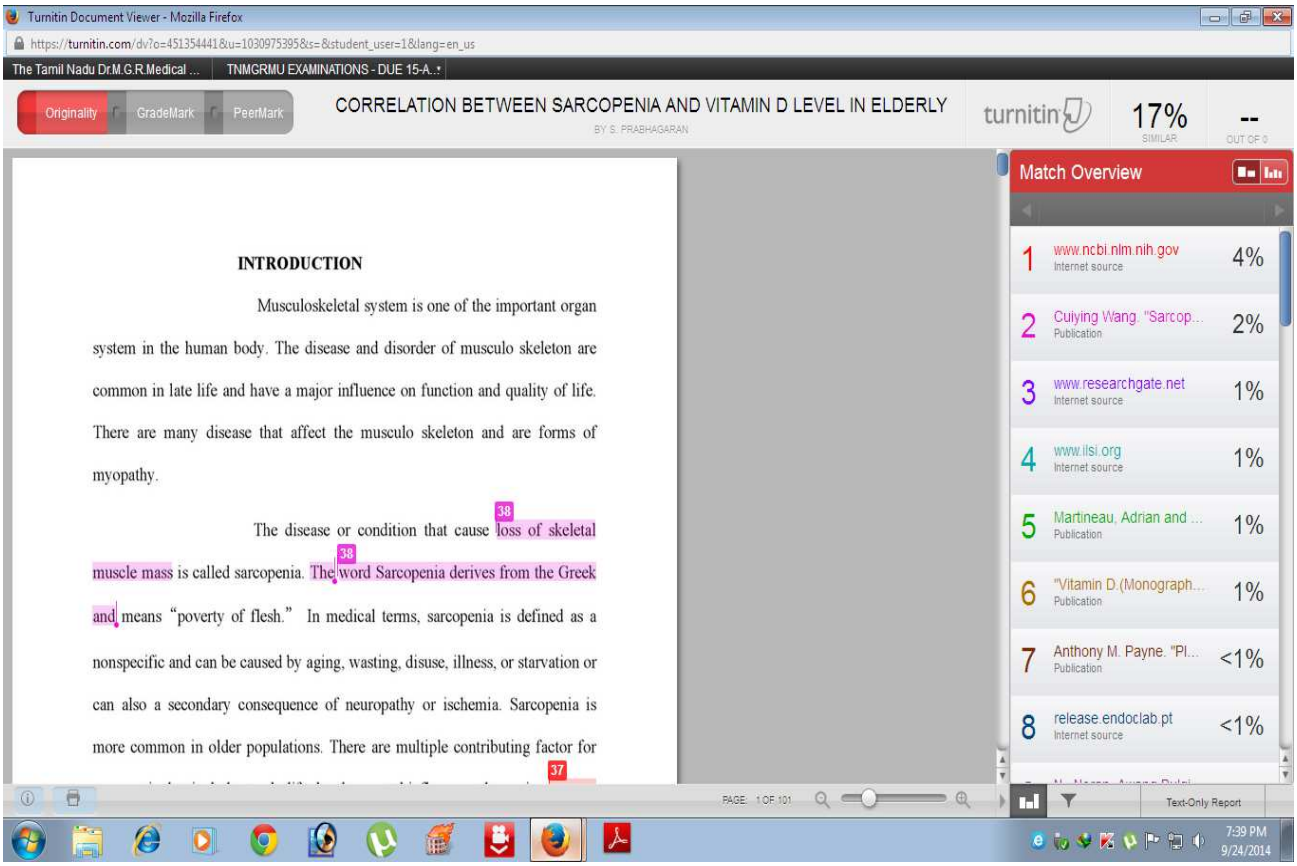
Sd/Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

Member Secretary, Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

TURNITIN PLAGIARISM SCREEN SHOT



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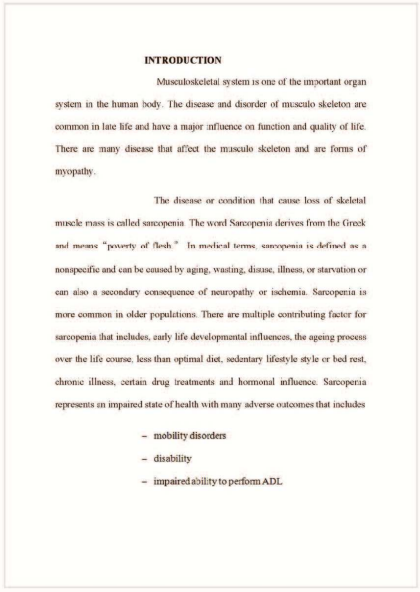

INTRODUCTION

Musculoskeletal system is one of the important organ system in the human body. The disease and disorder of musculo skeleton are common in late life and have a major influence on function and quality of life. There are many disease that affect the musculo skeleton and are forms of myopathy.

The disease or condition that cause loss of skeletal muscle mass is called sarcopenia. The word Sarcopenia derives from the Greek and means "poverty of flesh." In medical terms, sarcopenia is defined as a nonspecific and can be caused by aging, wasting, disease, illness, or starvation or can also a secondary consequence of neuropathy or ischemia. Sarcopenia is more common in older populations. There are multiple contributing factor for sarcopenia that includes, early life developmental influences, the ageing process over the life course, less than optimal diet, sedentary lifestyle style or bed rest, chronic illness, certain drug treatments and hormonal influence. Sarcopenia represents an impaired state of health with many adverse outcomes that includes

- mobility disorders
- disability
- impaired ability to perform ADL

ஆராய்ச்சி தகவல் தாள்

ஆராய்ச்சி தலைப்பு

முதியோர்களில் தசை வலிமை குறைந்தவர்களில் வைட்டமின்-டி அளவு மற்றும் அதன் தொடர்பு பற்றி ஆராய்ச்சி

ஆராய்ச்சியாளர் பெயர் : மரு.சு.பிரபாகரன்

ஆராய்ச்சி செய்யுமிடம் : முதியோர் பிரிவு, ராஜீவ் காந்தி அரசு பொது மருத்துவமனை, சென்னை

பங்கேற்பாளர் பெயர் : வயது : பாலினம்: ஆண் / பெண்

ஆராய்ச்சியின் நோக்கம்

முதியோர்களில் தசை வலிமை குறைந்தவர்களில் வைட்டமின்-டி அளவு மற்றும் அதன் தொடர்பு பற்றி ஆராய்வதே இந்த ஆராய்ச்சியின் நோக்கமாகும்.

ஆராய்ச்சி முறை

முதியோர்களின் தசை வலிமைக்கான பரிசோதனைகளான கைபிடிப்பு வலிமை, நடை வேகம் மற்றும் தசை திம்மை அளவு ஆகியவை செய்யப்படும். வைட்டமின்-டி அளவு தெரிந்துகொள்வதற்கு தங்களிடமிருந்து 2மி.லி. இரத்தம் எடுத்து இரத்தப்பரிசோதனை செய்து அதன் தொடர்பு பரிசோதனை செய்யப்படும்.

ஆராய்ச்சியின் பலன் மற்றும் தீங்கு

இந்த ஆராய்ச்சியின் மூலம் பங்கேற்பவருக்கு தீங்கு ஏதும் கிடையாது. நீங்களும் இந்த ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்புகிறோம்.

முடிவுகளை அல்லது கருத்துகளை வெளியிடும்போதோ அல்லது ஆராய்ச்சியின் போதோ தங்களது பெயரையோ அல்லது அடையாளங்களையோ வெளியிட மாட்டோம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த ஆராய்ச்சியின் முடிவுகளை ஆராய்ச்சியின்போது அல்லது ஆராய்ச்சியின் முடிவின் போது தங்களுக்கு அறிவிக்கப்படும் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இதனால் தங்களது ஆய்வறிக்கையோ, அன்றாட செயல்பாடுகளோ பாதிக்கப்படாது என்று தெரிவித்துக்கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின் பேரில் தான் இருக்கிறது. மேலும் நீங்கள் எந்நேரமும் இந்த ஆராய்ச்சியிலிருந்து பின்வாங்கலாம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

ஆராய்ச்சியாளர் கையொப்பம் **பங்கேற்பாளர் கையொப்பம்**

சு.பிரபாகரன்

நாள் :

இடம் :

INFORMATION SHEET

TITLE: CORRELATION BETWEEN SARCOPENIA AND VITAMIN D LEVEL IN ELDERLY POPULATION
NAME OF THE INVESTIGATOR: Dr.S.PRABHAGARAN

STUDY CENTRE: Dept. of Geriatric Medicine, Rajiv Gandhi Government General Hospital, Chennai.

NAME OF THE PARTICIPANT: **AGE:** **SEX:**

PURPOSE OF THE STUDY: The purpose of this study is to address the correlation between sarcopenia and vitamin D level in elderly population.

STUDY DESIGN: Observational Study

STUDY PROCEDURE: Patient will be selected according to the above mentioned criteria. Relevant history will be obtained. Anthropometric evaluation will be done. Skeletal muscle index will be evaluated using body composition analyzer. Muscle strength and muscle activity was analyzed using hand grip dynamometry and walking speed test respectively. 2ml of blood samples will be collected for estimating serum vitamin D level.

POSSIBLE RISKS: No possible risks by means of this study.

CONFIDENTIALITY OF THE INFORMATION OBTAINED FROM THE PATIENT: The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

DECISION TO PARTICIPATE IN THE STUDY: Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

RESULT OF THE STUDY:

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of Investigator **Dr.S.Prabagaran** Date : Place :	Signature of Participant

ஆராய்ச்சி ஒப்புதல் கடிதம்

ஆராய்ச்சி தலைப்பு

முதியோர்களில் தசை வலிமை குறைந்தவர்களில் வைட்டமின்-டி அளவு மற்றும் அதன் தொடர்பு பற்றி ஆராய்ச்சி

ஆராய்ச்சியாளர் பெயர்	:	மரு.சு.பிரபாகரன்
ஆராய்ச்சி செய்யுமிடம்	:	முதியோர் பிரிவு, ராஜீவ் காந்தி அரசு பொது மருத்துவமனை, சென்னை
பங்கேற்பாளர் பெயர்	:	
வயது	:	
பாலினம்	:	ஆண் / பெண்
அடையாள எண்	:	

இந்த ஆராய்ச்சின் விவரங்களும் அதன் நோக்கங்களும் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது.

எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்துகொண்டு எனது சம்மதத்தை தெரிவிக்கிறேன்.

எனக்கு முதியோர்களில் தசை வலிமை குறைந்தவர்களில் வைட்டமின்-டி அளவு மற்றும் அதன் தொடர்பு பற்றிய ஆராய்ச்சி செய்ய முழு சம்மதம்.

இந்த ஆராய்ச்சியில் பிறரின் நிர்பந்தமின்றி என் சொந்த விருப்பத்தின்பேரில் பங்கு பெறுகின்றேன். இந்த ஆராய்ச்சியில் இருந்து நான் எந்நேரமும் பின்வாங்கலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்துகொண்டேன்.

நான் இந்த ஆராய்ச்சியின் விபரங்களைக் கொண்ட ஆராய்ச்சித் தகவல் தாளைப் பெற்றுக் கொண்டேன்.

முதியோர்களின் தசை வலிமைக்கான பரிசோதனைகளான கைபிடிப்பு வலிமை, நடை வேகம் மற்றும் தசை திண்மை அளவு ஆகியவை செய்யப்படும். வைட்டமின்-டி அளவு தெரிந்துகொள்வதற்கு தங்களிடமிருந்து 2மி.லி. இரத்தம் எடுத்து இரத்தப்பரிசோதனை செய்து அதன் தொடர்பை அறிந்துகொள்வதே இந்த ஆராய்ச்சியின் நோக்கமாகும்.

இதன் மூலம் எந்த பின்விளைவும் ஏற்படாது என்று மருத்துவர் மூலம் தெரிந்து கொண்டு, நான் என்னுடைய சுய நினைவுடனும் மற்றும் முழு சுதந்திரத்துடனும் இந்த மருத்துவ ஆராய்ச்சியில் என்னை சேர்த்துக்கொள்ள சம்மதம் தெரிவிக்கிறேன்.

ஆராய்ச்சியாளர் கையொப்பம் **பங்கேற்பாளர் கையொப்பம்**

சு.பிரபாகரன்

நாள் :

இடம் :

PATIENT CONSENT FORM

Study Detail	:	**"Correlation between sarcopenia and vitamin D level in elderly population"**
Study Centre	:	Rajiv Gandhi Government General Hospital, Chennai.
Patient's Name	:	
Patient's Age	:	
Identification Number	:	

Patient may check (√) these boxes

a) I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction.	❑
b) I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected.	❑
c) I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study.	❑
d) I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms.	❑
e) I hereby consent to participate in this study.	❑
f) I hereby give permission to undergo complete clinical examination and hematological tests.	❑

Signature / thumb impression

Signature of Investigator

Patient's Name and Address :

Study Investigator's Name :

Dr. S.PRABHAGARAN

s#no	age	AGEGR	sex	HAND_GRIP	GAIT_SPEED_MSEC	weight	height	BMI	ms#_Mass_	IN_KG	SMI	vitD	group_0_no1yes2_severe	vitamingroup
1	77	>75 Yrs	Female	28	0.83	61	163	22.95909	31.5	19.215	7.232113	56.65	no	INSUFFICIENCY
2	75	<75 Yrs	Female	22	1.05	60	151	26.31464	25.5	15.3	6.710232	35.67	no	INSUFFICIENCY
3	72	<75 Yrs	Female	23	0.83	63	150	28	24.7	15.561	6.916	43.56	no	INSUFFICIENCY
4	66	<75 Yrs	Female	25	0.81	84	156	34.51677	23.8	19.992	8.21499	47.78	no	INSUFFICIENCY
5	65	<75 Yrs	Female	29	0.83	53	150	23.55556	27.3	14.469	6.430667	36.78	no	INSUFFICIENCY
6	73	<75 Yrs	Female	22	0.87	73	172	24.6755	27.7	20.221	6.835114	71	no	INSUFFICIENCY
7	70	<75 Yrs	Female	25	0.89	73	152	31.59626	22.1	16.133	6.982774	64.4	no	INSUFFICIENCY
8	75	<75 Yrs	Female	27	0.83	40	135	21.94787	31	12.4	6.803841	76.78	no	NORMAL
9	68	<75 Yrs	Female	22	0.94	66	158	26.43807	25.3	16.698	6.688832	64.67	no	INSUFFICIENCY
10	65	<75 Yrs	Female	26	1.05	49	142	24.30073	26.8	13.132	6.512597	51.34	no	INSUFFICIENCY
11	66	<75 Yrs	Female	26	0.86	56	150	24.88889	26.6	14.896	6.620444	55.75	no	INSUFFICIENCY
12	70	<75 Yrs	Female	25	0.85	75	151	32.89329	23.6	17.7	7.762817	84.56	no	NORMAL
13	77	>75 Yrs	Female	23	1.02	57	153	24.34961	27.3	15.561	6.647443	48.05	no	INSUFFICIENCY
14	68	<75 Yrs	Female	25	0.82	59	142	29.26007	26.2	15.458	7.666138	54.45	no	INSUFFICIENCY
15	72	<75 Yrs	Female	27	0.83	81	155	33.71488	19.7	15.957	6.641831	22.13	no	DEFICIENCY
16	67	<75 Yrs	Female	28	0.97	49	146	22.98743	28.5	13.965	6.551417	83.34	no	NORMAL
17	68	<75 Yrs	Female	26	0.94	80	153	34.17489	19.8	15.84	6.766628	61.17	no	INSUFFICIENCY
18	65	<75 Yrs	Female	26	0.92	56	148	25.56611	26.7	14.952	6.82615	65.36	no	INSUFFICIENCY
19	65	<75 Yrs	Female	23	0.92	50	140	25.5102	27.2	13.6	6.938776	85.46	no	NORMAL
20	75	<75 Yrs	Female	23	0.97	55	154	23.1911	29.7	16.335	6.887755	61.21	no	INSUFFICIENCY
21	73	<75 Yrs	Female	27	1	53	145	25.20809	28.7	15.211	7.234721	60.45	no	INSUFFICIENCY
22	70	<75 Yrs	Female	24	0.85	60	154	25.29938	26.2	15.72	6.628436	45.45	no	INSUFFICIENCY
23	67	<75 Yrs	Female	26	0.83	41	144	19.77238	34.2	14.022	6.762153	66.32	no	INSUFFICIENCY
24	66	<75 Yrs	Female	28	0.98	51	150	22.66667	31.1	15.861	7.049333	70.13	no	INSUFFICIENCY
25	73	<75 Yrs	Female	23	1.12	51	148	23.28342	28.6	14.586	6.659058	24.83	no	DEFICIENCY
26	66	<75 Yrs	Male	38	0.87	67	171	22.91303	42.2	28.274	9.6693	85.67	no	NORMAL
27	73	<75 Yrs	Male	33	0.85	60	161	23.14726	38.8	23.28	8.981135	51.37	no	INSUFFICIENCY
28	80	>75 Yrs	Male	36	0.86	67	171	22.91303	39.5	26.465	9.050648	65.65	no	INSUFFICIENCY
29	72	<75 Yrs	Male	38	0.85	65	153	27.7671	32.7	21.255	9.079841	86.44	no	NORMAL
30	77	>75 Yrs	Male	34	0.84	67	160	26.17188	33.9	22.713	8.872266	48.76	no	INSUFFICIENCY

31	67	<75 Yrs	Male	34	0.81	74	158	29.64269	32.5	24.05	9.633873	51.34	no	INSUFFICIENCY
32	65	<75 Yrs	Male	50	0.82	79	174	26.09328	34.8	27.492	9.08046	46.45	no	INSUFFICIENCY
33	66	<75 Yrs	Male	38	0.83	77	170	26.6436	33.3	25.641	8.872318	79.1	no	NORMAL
34	68	<75 Yrs	Male	37	0.92	83	173	27.7323	35.2	29.216	9.76177	75.87	no	NORMAL
35	78	>75 Yrs	Male	36	0.89	78	163	29.35752	31.3	24.414	9.188904	62.2	no	INSUFFICIENCY
36	77	>75 Yrs	Male	36	0.88	65	148	29.67495	30	19.5	8.902484	48.8	no	INSUFFICIENCY
37	76	>75 Yrs	Male	42	0.81	77	154	32.46753	29	22.33	9.415584	42.65	no	INSUFFICIENCY
38	75	<75 Yrs	Male	38	0.82	75	149	33.78226	26.8	20.1	9.053646	41.45	no	INSUFFICIENCY
39	75	<75 Yrs	Male	44	0.85	84	169	29.41074	32.5	27.3	9.558489	56.65	no	INSUFFICIENCY
40	79	>75 Yrs	Male	38	0.92	77	165	28.28283	35	26.95	9.89899	80.01	no	NORMAL
41	70	<75 Yrs	Male	50	1.12	87	164	32.34682	28	24.36	9.057109	55.67	no	INSUFFICIENCY
42	66	<75 Yrs	Male	38	0.85	66	148	30.13148	31.2	20.592	9.401023	43.56	no	INSUFFICIENCY
43	74	<75 Yrs	Male	34	0.86	76	162	28.959	31.6	24.016	9.151044	77.78	no	NORMAL
44	68	<75 Yrs	Male	50	0.86	69	151	30.26183	31.3	21.597	9.471953	56.12	no	INSUFFICIENCY
45	70	<75 Yrs	Male	46	0.87	61	153	26.05835	35	21.35	9.120424	23.78	no	DEFICIENCY
46	74	<75 Yrs	Male	50	0.91	69	164	25.65437	34.6	23.874	8.876413	77.89	no	NORMAL
47	65	<75 Yrs	Male	38	0.84	74	165	27.1809	33.6	24.864	9.132782	61.34	no	INSUFFICIENCY
48	69	<75 Yrs	Male	38	0.83	86	162	32.7694	27.9	23.994	9.142661	54.45	no	INSUFFICIENCY
49	72	<75 Yrs	Male	36	0.91	59	156	24.24392	37.7	22.243	9.139957	54.4	no	INSUFFICIENCY
50	80	>75 Yrs	Male	50	0.92	65	162	24.76757	36.8	23.92	9.114464	69.99	no	INSUFFICIENCY
1	78	>75 Yrs	Female	3	0.47	42	149	24.5	18.5	7.77	3.499842	24.5	Severe	DEFICIENCY
2	75	<75 Yrs	Female	3	0.7	46	151	26.54	18.7	8.602	3.772642	26.54	Severe	INSUFFICIENCY
3	80	>75 Yrs	Female	2	0.58	37	134	34.56	16	5.92	3.296948	34.56	Severe	INSUFFICIENCY
4	75	<75 Yrs	Female	13	0.43	34	147	28.05	17.8	6.052	2.800685	28.05	Severe	INSUFFICIENCY
5	75	<75 Yrs	Female	2	0.33	35	138	76.45	16.2	5.67	2.977316	76.45	Severe	NORMAL
6	74	<75 Yrs	Female	3	0.48	44	144	45.32	17.5	7.7	3.713349	35.32	Severe	INSUFFICIENCY
7	70	<75 Yrs	Female	12	0.43	33	138	30.13	18.1	5.973	3.136421	30.13	Severe	INSUFFICIENCY
8	70	<75 Yrs	Female	3	0.64	37	152	42.15	21.5	7.955	3.443127	42.15	Severe	INSUFFICIENCY
9	75	<75 Yrs	Female	1	0.76	35	139	43.34	15.7	5.495	2.844056	33.34	Severe	INSUFFICIENCY
10	80	>75 Yrs	Female	11	0.68	43	153	23.45	21.1	9.073	3.87586	23.45	Severe	DEFICIENCY
11	77	>75 Yrs	Female	2	0.84	44	146	41.17	18.7	8.228	3.860011	41.17	Severe	INSUFFICIENCY
12	76	>75 Yrs	Female	1	0.76	44	153	35.36	19	8.36	3.571276	35.36	Severe	INSUFFICIENCY
13	78	>75 Yrs	Female	1	0.65	46	146	25	17.3	7.958	3.733346	25	Severe	DEFICIENCY
14	76	>75 Yrs	Female	8	0.78	40	141	77.75	16.4	6.56	3.299633	31.72	Severe	INSUFFICIENCY
15	70	<75 Yrs	Female	8	0.64	39	149	44.56	20.3	7.917	3.566056	44.56	Severe	INSUFFICIENCY

16	80	>75 Yrs	Female	22	0.7	38	145	37.56	16.6	6.308	3.000238	31.21	Mild	INSUFFICIENCY
17	67	<75 Yrs	Female	26	0.51	43	135	31.21	16.3	7.009	3.845816	32.45	Mild	INSUFFICIENCY
18	68	<75 Yrs	Female	13	1.2	36	143	32.45	19.3	6.948	3.397721	77.75	Mild	NORMAL
19	70	<75 Yrs	Female	24	0.42	42	145	31.72	17.4	7.308	3.475862	51.45	Mild	INSUFFICIENCY
20	74	<75 Yrs	Female	13	0.9	39	138	31.45	19.1	7.449	3.911468	79.98	Mild	NORMAL
21	78	>75 Yrs	Female	25	0.54	43	153	79.98	21.4	9.202	3.930967	24.98	Mild	DEFICIENCY
22	69	<75 Yrs	Female	8	0.9	37	139	24.98	20.4	7.548	3.90663	36.67	Mild	INSUFFICIENCY
23	70	<75 Yrs	Female	2	0.83	49	153	36.67	18.6	9.114	3.893374	35.45	Mild	INSUFFICIENCY
24	75	<75 Yrs	Female	26	0.55	38	141	35.45	18.9	7.182	3.612494	41.21	Mild	INSUFFICIENCY
25	70	<75 Yrs	Female	16	1	33	144	41.21	16.6	5.478	2.641782	37.56	Mild	INSUFFICIENCY
26	69	<75 Yrs	Male	14	0.83	53	161	80.13	24.1	12.773	4.927665	80.13	Mild	NORMAL
27	75	<75 Yrs	Male	18	0.87	47	153	41.45	21.4	10.058	4.296638	41.45	Mild	INSUFFICIENCY
28	73	<75 Yrs	Male	12	0.85	52	157	76.34	23.4	12.168	4.936509	76.34	Mild	NORMAL
29	75	<75 Yrs	Male	18	0.82	54	168	78.83	28.6	15.444	5.471939	78.83	Mild	NORMAL
30	75	<75 Yrs	Male	10	0.81	54	173	27.24	27.8	15.012	5.015871	27.24	Mild	INSUFFICIENCY
31	77	>75 Yrs	Male	24	0.87	57	163	36.76	25.3	14.421	5.427754	36.76	Mild	INSUFFICIENCY
32	76	>75 Yrs	Male	22	0.83	53	167	33.33	26.7	14.151	5.074044	33.33	Mild	INSUFFICIENCY
33	76	>75 Yrs	Male	16	0.91	64	162	36.56	22.6	14.464	5.511355	36.56	Mild	INSUFFICIENCY
34	78	>75 Yrs	Male	22	0.81	55	155	47.89	23.6	12.98	5.402706	47.89	Mild	INSUFFICIENCY
35	76	>75 Yrs	Male	6	0.81	49	171	76.32	28.5	13.965	4.775828	76.32	Mild	NORMAL
36	75	<75 Yrs	Male	6	0.83	51	160	36.56	24.3	12.393	4.841016	36.56	Mild	INSUFFICIENCY
37	78	>75 Yrs	Male	20	0.86	50	159	25.67	24.6	12.3	4.865314	25.67	Mild	INSUFFICIENCY
38	80	>75 Yrs	Male	6	0.58	44	156	34.79	21.8	9.592	3.941486	56.34	Severe	INSUFFICIENCY
39	78	>75 Yrs	Male	13	0.7	50	157	56.34	23.4	11.7	4.746643	44.44	Severe	INSUFFICIENCY
40	78	>75 Yrs	Male	13	0.3	39	158	44.44	25.8	10.062	4.030604	41.37	Severe	INSUFFICIENCY
41	78	>75 Yrs	Male	14	0.69	47	153	41.37	22.8	10.716	4.577727	33.34	Severe	INSUFFICIENCY
42	75	<75 Yrs	Male	7	0.4	57	167	33.34	25.8	14.706	5.273047	42.11	Severe	INSUFFICIENCY
43	77	>75 Yrs	Male	13	0.74	43	153	42.11	21.4	9.202	3.930967	25.65	Severe	INSUFFICIENCY
44	78	>75 Yrs	Male	4	0.35	41	151	25.65	22.6	9.266	4.063857	35.11	Severe	INSUFFICIENCY
45	76	>75 Yrs	Male	6	0.72	38	146	35.11	22.3	8.474	3.975418	29.85	Severe	INSUFFICIENCY
46	71	<75 Yrs	Male	20	0.75	47	161	29.85	27.9	13.113	5.058833	36.54	Severe	INSUFFICIENCY
47	73	<75 Yrs	Male	24	0.75	58	168	36.54	24.9	14.442	5.116922	24	Severe	DEFICIENCY
48	80	>75 Yrs	Male	28	0.65	46	148	24	20	9.2	4.200146	46.44	Severe	INSUFFICIENCY
49	80	>75 Yrs	Male	14	0.64	51	155	46.44	22.7	11.577	4.81873	23.5	Severe	DEFICIENCY
50	72	<75 Yrs	Male	24	0.4	50	170	23.5	28.4	14.2	4.913495	34.79	Severe	INSUFFICIENCY